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1. food poisoning (3) infection

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9 " fish (12) food allergy

13. botulism (14) canned foods

15. laboratory investigation

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FOOD POISONING

FOOD POISONING

FOOD-BORNE INFECTION AND INTOXICATION

Nature, history, and causation
Measures for prevention and control

Fourth Edition

by ELLIOT B. DEWBERRY, M.B.E.

Fellow of the Royal Society of Health, Fellow of the Royal Institute of Public Health and Hygiene

Foreword by

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DEDICATED

TO THE MEMORY OF

JOHN ROSE

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ABERDEEN

FOREWORD

ɨood wine needs no bush', and Mr. Dewberry's book has already roved its value as a standard work. At the same time, I welcome ıe opportunity of emphasizing that this fourth edition is practi-ılly a new book, for the author with access to further sources of ıformation has largely rewritten it. In doing so he has drawn pon the recent findings of bacteriologists, epidemiologists, ɔxicologists, and medical officers of health, and has assembled his ɔllated facts in a readable and succinct form.

From earliest times mankind must have realized the poisonous ature of certain foods and waters, and taken precautions against ıeir effects. The possibility of 'death in the pot', as recorded in Kings iv, was known before the times of the sons of the prophets. 'he interest of this book is heightened by an account of the history f food poisoning and of the pioneers who investigated its aetiology.

In spite of increased knowledge of the subject as, for example, ir William Savage's comprehensive account of the salmonella ıfections, the increase in communal feeding both during and since ıe Second World War has favoured food-poisoning outbreaks. In 955 the incidence of such outbreaks in England and Wales was 9 per cent above that of the year before. In that year also, for ıe first time for many years, two cases of botulism were reported.

Processed or made-up meat dishes are responsible for a high roportion of the outbreaks of food poisoning. The incidence of ıose outbreaks reflects failure to apply measures of prevention hose value has already been demonstrated. Strict cleanliness in ıe preparation and handling of food is essential.

This fourth edition of Mr. Dewberry's book is therefore timely. includes many important facts and constitutes a valuable work reference for all interested in this vital problem of public health.

ARTHUR MACNALTY

AUTHOR'S PREFACE TO FIRST EDITION

In compiling this work, my intention has been to collect and present in readable form in one volume the fundamental facts relative to the many kinds of human food poisoning. The selection of essential material has been somewhat difficult, because a very large part of the information on the various subjects, especially bacterial food poisoning, is so widely distributed in numerous medical works, scientific treatises, journals and pamphlets, or recorded in Public Health Reports published during the past decade as a result of the investigations, studies and experiments by medical experts and observers in this country, the Colonies and the Public Health services and Universities in the United States of America.

No originality is claimed for this book. Many well-known works of reference have been consulted, and I gratefully acknowledge my indebtedness to the authors concerned.

Some interesting historical matter concerning early food-poisoning investigations has been included to indicate the sequence of events leading up to important bacteriological discoveries.

References are appended to each chapter for the use of readers desirous of consulting the original articles or books.

Quotations and Figures 28, 29, 30, 32 and 33 from official publications are included by permission of the Controller of His Majesty's Stationery Office, the Ministry of Agriculture and Fisheries and the Ministry of Health.

An Appendix on the Contamination (and Decontamination) of Foods by Poisonous Gases used in War has been kindly contributed by Mr. Henry Eastwood, M.R.SAN.I., Food Contamination Officer, Borough of Hornsey, London.

I am greatly indebted to Sir William Savage, M.D., for his valuable assistance, and my sincere thanks are accorded to Professor W. W. C. Topley and Professor G. S. Wilson for kindly permitting me to quote from their work on *The Principles of Bacteriology and Immunity*; to friends, both at home and abroad, including Professor K. F. Meyer, Dr. J. G. Geiger, Dr. F. W. Tanner and Dr. S. R. Damon, for allowing me to make extracts from their writings, and to the Rockefeller Institute for Medical

Research, New York, for consenting to excerpts being reprinted from their monograph, *Botulism* by the late Dr. Ernest Dickson.

I am grateful, too, to all those who loaned photographs of some of the early investigators and to the publishers of *Food Manufacture*, for their guidance and help. Finally, I must acknowledge the valuable help received from my wife in the preparation of the manuscript and index.

E. B. D.

Epsom, 1943

PREFACE TO SECOND EDITION

It is gratifying to note that the demand for this work has necessitated the publication of a second edition, the preparation of which has afforded an opportunity of carefully revising certain parts of the text, and of incorporating new and up-to-date material, including a chapter on staphylococcus food poisoning. This particular type of illness, due to enterotoxin-producing staphylococci, has excited considerable interest and discussion. Investigations and much experimental work have been carried out in this connection during the past few years.

My sincere thanks are accorded to Professor C. E. Dolman for kindly permitting me to quote from his writings on this subject.

A new Appendix has been added on the laboratory investigation of food poisoning cases and the media recommended for the isolation of members of the salmonella group. I am extremely grateful to Dr. J. E. McCartney for his valuable help and suggestions in its preparation.

Additional photographs have been included in order to add interest to the work, which it is hoped will be received as favourably as its predecessor.

The reproductions of poisonous fungi are from Bulletin No. 23, Ministry of Agriculture and Fisheries, by permission of the Controller of His Majesty's Stationery Office.

E. B. D.

Epsom, 1946

PREFACE TO THIRD EDITION

In this, the third edition of *Food Poisoning*, every endeavour has been made to incorporate the latest information, consequently, several chapters have been partly or wholly re-written.

Recently, considerable prominence has been given in the technical press to Ice-cream, Food Handlers, and Food Hygiene. Information on these important subjects is embodied.

Acknowledgements—Except for a few minor alterations, the frontispiece diagram of the various Animal Reservoirs of Food and Drink Infections was prepared by the Central Public Health Laboratories for the Central Council for Health Education.

Thanks to the United States Public Health Reports, an Appendix dealing with gastro-enteritis aboard ship is included.

Extracts from the Annual Reports of the Chief Medical Officer of the Ministry of Health are reproduced by permission of the Controller of His Majesty's Stationery Office.

My sincere thanks are accorded to friends at home and abroad for permitting me to quote from their valuable articles and writings and to cite interesting cases recorded by them.

E. B. D.

Epsom, 1950

PREFACE TO FOURTH EDITION

It is gratifying to note that the demand for this work has necessitated the publication of a fourth edition. The text has been revised and every endeavour made to incorporate the latest information on the subjects. Chapters on food hygiene and inspection and on canned foods have been contributed by Cecil Ash, Fareham, U.D.C., whose valuable assistance with the preparation of other chapters in the work is gratefully acknowledged. The number of references has been increased for the convenience of readers desirous of consulting the original articles or books quoted. Additional photographs are included in order to add interest to the work, which it is hoped will be received as favourably as its predecessor.

My sincere thanks are accorded to friends and colleagues at home and abroad for kindly granting me permission to quote from their articles or writings or to cite cases and outbreaks of food poisoning recorded by them, and also for the loan of interesting photographs from Dr. Bruce Halstead and Dr. W. M. Lively, Jr., of poisonous tropical fish; Dr. Stuart Mudd, Dr. Robley Williams, and Dr. Van Iterson for micrographs of organisms; Messrs. Reads Ltd., Liverpool, and the American Can Company, New York, for those of early food canning operations.

Many well-known books of reference have been consulted and I acknowledge my indebtedness to the authors concerned. My sincere thanks to Sir William Savage, M.D., for his valuable suggestions and advice and to Dr. J. E. McCarthy for so kindly allowing excerpts to be made from his *Handbook of Bacteriology*. Extracts from the Annual Reports of the Chief Medical Officer to the Ministry of Health are reproduced by kind permission of the Controller of Her Majesty's Stationery Office.

Finally I must acknowledge the valuable help received from my wife in the preparation of the manuscript.

E. B. D.

Epsom, 1958

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PART I

Chapter I

INTRODUCTION

THE term 'Food Poisoning', used in its broadest sense, embraces a variety of human ailments caused by poisonous substances transmitted by the food or drink ingested. In its strictly technical sense, however, it is confined to infections and intoxications associated with certain pathogenic organisms; the majority of outbreaks today are of this type.

Food-poisoning outbreaks frequently occur during the summer months. Sometimes they assume large proportions, especially when the milk supply is the source of infection. Owing to a considerable number of outbreaks being of a mild and temporary type and limited to one or more persons or members of the family, they are frequently overlooked or are not investigated. Only when the malady is of a really serious nature and medical advice is sought, or when a considerable number of persons are attacked simultaneously are enquiries made into the origin. Since food poisoning was made notifiable, and greater facilities for bacteriological examination and researches became available, more cases and outbreaks are scientifically investigated. It is absolutely necessary that all investigations be carried out without delay, for team-work and speed are essential; otherwise much useful information may be lost. Moreover, early enquiries also enable any necessary precautionary measures to be instituted, including the collection of suspected foods and specimens, etc., for chemical analysis or bacteriological examination. There are, of course, certain other illnesses where the symptoms (vomiting and gastro-intestinal disturbance) closely resemble those of food poisoning.

To ptomaines have been assigned the chief cause, not only of the harmful effects resulting from the ingestion of tainted meats, but of food poisoning generally, and in consequence it has been difficult to eradicate the indiscriminately applied term 'ptomaine' poisoning. Proof has been definitely established that these putrefactive alkaloids are not present in the early stages of decomposition and are only formed when putrefaction has advanced to such a degree that the food becomes repulsive.

Substantiation of this is to be found in an address given before the London Chamber of Commerce by the late Sir William Wilcox, who said:

> The idea that food poisoning is due to ptomaines is quite exploded. I have made a very large number of analyses in fatal cases of poisoning and suspected poisoning; but although I searched most minutely for all signs of alkaloidal poisons, ptomaines, and so on, unless there are some genuine chemical poison there, my efforts to find these poisons failed. I used not to succeed in finding ptomaines in the viscera which were examined, though many of them were of an extremely advanced nature as regards decomposition which had occurred. So that we can dismiss these ptomaines as the cause of food poisoning.

When meat and meat foods become stale and decompose, certain chemical and bacteriological changes occur. These are caused by enzymes present in small quantities in the meat and by putrefactive organisms. The enzymes are responsible for breaking down the proteins into simpler substances and, in consequence, the physical appearance of the food may become altered. Later, oxidation of the fats takes place and unpleasant flavours and odours are produced. The putrefactive organisms, however, are the chief products of stale and decomposed food. Although these organisms are not harmful they sometimes tend to outgrow and so mask any pathogenic bacteria that may be present in contaminated food. Such food might, if consumed, give rise to food poisoning.

Researches in bacteriology and pathology furnish conclusive proof that the majority of cases of food poisoning (apart from non-bacterial food poisoning) are due to infection of the human subject by pathogenic organisms, or to the toxins they manufacture. The term 'ptomaine' poisoning used in connection with food poisoning, therefore, is misleading and should be discarded in all scientific literature.

The provision of an attractive uncontaminated and unadulterated food supply is a problem of vital importance and one that has never excited so much interest in the medical profession, Government departments, public health officials, educational authorities and food manufacturers as it has during the past few years. Food is now prepared, preserved and manufactured in immense quantities by various methods and processes, often by massed production. Machinery has to a large extent replaced manual labour; this entails close supervision and the problem of effectively cleaning and sterilizing all parts of the machinery which may come into contact with the foodstuffs, in order to avoid risk of

contamination. Food products are frequently transported long distances in a variety of vehicles under varying conditions and are handled by a considerable number of persons before finally reaching the consumer. Thus they are exposed to contamination of all descriptions through carelessness or ignorance.

In recent years, however, there has been an important metamorphosis. The major portion of our food supply has been beyond criticism or suspicion. This is attributable not merely to legislation, which exacts in every way higher standards for products and manufacture, but to a genuine desire on the part of manufacturers, canners and traders to place on the market clean, wholesome food. Through their various trade organizations, by bacteriological and chemical research and other means, marked progress has been made in manufacture, preservation, storage, transportation and distribution. Control of bacteria in food is now the aim of a large number of industries. This is accomplished by such means as pasteurization, processing, the use of harmless preservatives, refrigeration, quick freezing, etc. The safeguarding and controlling of our food supplies goes to the very root of public health, and it is only by investigation and elucidation of the many difficulties associated with food poisoning, as briefly referred to above and amplified at some length in this work, that we have been able to make material and satisfactory progress towards the solution of a big problem, fraught as it is with innumerable complexities.

Statistics reveal that in England and Wales during the past few years there has been a continuous and steady increase in the number of recorded cases of food poisoning. This may be due in a measure to several contributing causes. Among them may be mentioned the changing food habits of persons of all ages and in all walks of life and consequent increase in the numbers who now take their meals away from home in restaurants, lunch clubs, canteens, cafeterias, snack and milk bars, schools, service camps, and training centres, etc., where any defects in hygienic control may affect a considerable number of adults and children.

Wilson (1955) points out that:

> In the communal kitchens practices have been taken over from the home which, though unobjectionable when applied to small quantities of food, present dangers of their own when large masses of food are being handled. In addition, a great variety of foods, cooked and uncooked, processed and unprocessed, contaminated to some degree with potentially dangerous organisms, have been coming on to the market and causing not only food poisoning but a wide dissemination of infective materials among the human and animal population.

Communal feeding also adds to the risk of food-borne disease since infected persons may contaminate food and utensils and so spread the infection.

Considerable difficulties have been experienced in commercial catering establishments consequent upon the shortage of trained personnel, resulting in lack of individual and general hygiene, such as the indiscriminate and unnecessary handling of food; infrequent attention to personal cleanliness; inefficient cleansing and sterilization of crockery-ware; the use of unclean cooking and food utensils, cracked and chipped cups, and other drinking vessels.

There has been lack of washing facilities (lavatory basins and adequate supplies of hot water, towels, and soap), W.C. and urinal accommodation for the staff conveniently near their work. In addition, there has been insufficient equipment such as refrigerators; suitable and efficient dish-washing machines; inadequate vermin-proof storage accommodation, and overburdening of kitchen facilities. Doubtless, enforcement of the Food Hygiene Regulations, 1955 will gradually improve the hygiene (personal and general) and sanitary conditions of all food premises, etc.

Regarding personnel in food establishments, it is clear that apart from legislation, the compulsory and adequate training and efficient education of such persons in all matters pertaining to the preparation and handling of food is essential, as well as their constant supervision when actually employed at their duties.

Other factors contributing to the increase are: (*a*) the importation into this country of frozen, liquid, and dried whole egg, albumin or yolk, used extensively in the confectionery industry, which on occasions has been found to be infected with harmful organisms; and (*b*) the increase in the number of human symptomless carriers of salmonellae.

REFERENCE

Wilson (1955): *J. Appl. Bact.*, **18**, 629.

Chapter II

HISTORICAL

FROM time immemorial food has been recognized as a cause of disease. Down through the ages man has gained considerable knowledge—often unpleasant or painful—as to what is fit and what is not fit to eat. Only in comparatively recent years have investigations been made and definite information obtained as to the origin and nature of the disease-producing properties associated with certain foods.

Meat, frequently the cause of outbreaks of illness, was used as an article of diet from the earliest times. Researches of geologists prove that prehistoric man lived partly on the flesh of animals. In Biblical times (Leviticus xi. 39) Moses commanded the Israelites not to eat the meat of animals affected with wasting diseases. In chapters xii and xv of Leviticus actual details are given as to what constituted being unclean both in person and clothing. The washing of the hands after touching anything unclean, particularly before meals, is emphasized in both the Old and New Testaments. The higher hieroglyphics of the Egyptians revealed that meat and meat foods entered largely into the dietary of the ancient nations and regulations regarding their use were officially enforced. They were forbidden to eat pork because it produced an excess of humours and eruptions. The Phoenicians had similar codes to the Egyptians regarding the consumption of animal flesh. Even in those far-off days it was recognized that animals which had died a natural death, or were killed 'to save their lives' were unfit for human food.

Food poisoning mentioned in the ancient writings of Hippocrates (460 B.C.), Horace (65 B.C.), Ovid (43 B.C.) and other writers and philosophers was of a somewhat different nature; it resulted from the accidental consumption of poisonous fungi, herbs and plants.

Records tell us that the Greek poet Euripides (480 B.C.) lost his wife, daughter and two sons, who during his absence had eaten poisonous fungi in mistake for the edible variety.

Theophrastus (300 B.C.), in his history of plants, makes several references to poisons, and records that these were sometimes added to food with criminal intent or for monetary greed. Zenophon

(400 B.C.) remarks that the addition of poison to food and drink was so common amongst the Medes that it was customary for the cup-bearers to taste the wine before it was offered to the king. In the Middle Ages intentional poisoning was so common that official food tasters were appointed.

During the Roman period oysters were used by Empresses, who were not the most devoted or virtuous of wives, as easy and agreeable agents in which to administer poison to their husbands or lovers. The Romans consumed pork, but the meat of goats was regarded as unclean.

Historical records mention a number of interesting incidents in which food was adulterated in Roman and Greek times.

In England during the early Middle Ages adulteration of food was practised with impunity. Sick animals were slaughtered and the diseased meat disguised or treated with preservatives and sold as sound food; the result can be well imagined. Cleanliness in slaughter-houses and premises where food was prepared was unheard of.

In 1319, the wardens of the City of London condemned two carcases of beef as being 'putrid and poisonous'. The would-be purveyor was convicted by a jury of attempting to sell 'bodies that have died of disease'. He was pilloried, and the offending carcases were burnt under his nose (Drummond and Wilbraham, 1958). Regulations in the fourteenth and fifteenth centuries forbade the use of tainted meat in public cook-shops which had been established on the bank of the river Thames.

During the early part of the nineteenth century investigators of cases of food poisoning (especially meat) assigned their cause to chemical poisons in decomposed food; later, however, they were attributed to putrefactive alkaloids (ptomaines). Such outbreaks were not associated with any bacterial theories.

Albert von Haller made the first scientific observations and experiments relating to the effects of decomposed protein substances upon animals. He injected aqueous extracts of putrid meat and blood into their circulations, which caused symptoms resembling those seen in septic diseases. Experimental work on these lines was also carried out by Gaspard (1822–4) and Magendies (1823) and aroused great interest.

Panum (1856), a Kiel professor, attempted to disclose the nature of the septic poison. He demonstrated that the poisonous qualities exhibited by putrid fish were of a chemical nature and undestroyed by boiling. Bergmann and Schmiedeberg (1868)

believed that the active poison was a substance they termed 'sepsin'. Later, more extensive studies were made upon the poisons in decomposed food, especially putrefying meat, and upon their effects on animals. This resulted in the publication of voluminous literature on the subject, amongst which were the monographs by Hiller (1879) and Gussenbauer (1882). Putrefactive alkaloids designated 'ptomaines' by Francesco Selmi (1872), the Italian chemist, were isolated by Nencki in 1876. In 1882–9, Brieger, Ladenburg, Vaughan, and Novy investigated these substances and found they possessed highly poisonous properties, especially when injected into animals. Ladenburg (1883) prepared the first putrefactive alkaloid (Cadaverine) by synthetic methods, and in 1888 Vaughan and Novy compiled a work on ptomaines and leucomaines. Vaughan (1884) isolated 'tyrotoxicon' (a substance closely allied to ptomaines) from cheese, which had caused symptoms of poisoning.

The ptomaine theory, although it at times caused considerable controversy amongst scientists and the medical profession, was nevertheless widely accepted for many years, and the general presumption was that the real cause of food poisoning had been discovered.

It may be mentioned in passing that it was suggested by Schwaun (1837) that putridity was really a biological process; this was confirmed by Pasteur in 1863.

The works of Vaillard (1902), Fornario (1906), Cathcart (1906) and other observers have since proved that these substances were comparatively non-toxic to experimental animals except when administered in excessively large doses, far larger than ever likely to be ingested under natural conditions. Also that ptomaines were not present in food until it had reached an advanced stage of decomposition when it would be repugnant in appearance and nauseating to the normal senses. Moreover, cases of food poisoning often resulted from the consumption of meat which showed no sign of decomposition and was normal in appearance.

Savage (1921) studied the relation of putrid food to illness. This was his opinion on the subject:

> The view which credits decomposed food with toxic properties largely rests upon a misconception due to the isolation of non-specific poisonous bodies called ptomaines from decomposing food, and then assuming that these bodies which are toxic by ingestion, and not at all, or to a very limited extent, by feeding, are the cause of food poisoning. . . . I have fed a series of kittens with extremely putrid mixtures of canned meat and fish over long periods and without demonstrating any

definite signs of toxicity. I am unaware of, and have been quite unable to find, any evidence in favour of the popular conception as to the great toxicity of incipiently putrid food or even definitely decomposed food; . . . there is no evidence of any scientific value that the general public runs any risk of illness from this source.

It is extremely unlikely, except under the conditions which existed in some concentration camps during the world wars, that human beings would be compelled to consume any food in an advanced stage of decomposition and in sufficient quantity to cause illness.

Tanner (1933) summarized the objections to ptomaine poisoning as a cause of illness as follows:

(1) Foods which would cause it would have to be in the later stages of decomposition, since presence of ptomaines is related to putrefaction. Most people would refuse to partake of such food.

(2) Some foods are purposely putrefied in order to improve their flavour. Such is the case with cheese, and even with meat, although in the latter case it is not carried as far as in the former. The Chinese also allow eggs to age.

(3) The toxicity of ptomaines isolated from putrefied foods has not been satisfactorily established.

(4) Symptoms of ptomaine poisoning are too inconclusive and resemble those caused by toxins formed, for instance, by members of the Salmonella group.

(5) Investigation of outbreaks of illness at first supposed to have been caused by ptomaines, has revealed more satisfactory explanations (botulism, Salmonella, toxins, etc.).

(6) If ptomaines were responsible for illness, many of us would be ill much of the time. It would be difficult to avoid foods which did not contain ptomaines as they are now conceived in the minds of many.

Among the early records of cases of meat poisoning in England Mackey (1873) describes a small outbreak which occurred at Hampton-in-Arden, Warwickshire, following the consumption of pork brawn. Sixteen persons (men, women, and children) were attacked with vomiting, violent gastro-intestinal disturbance, muscular cramps, and constriction of the throat, about 2 to 3 hours after eating the food. No deaths occurred and all were convalescent the next day. The brawn was purchased from one provision dealer in the village. He had eaten a small amount of the food without any harmful effects and could give no reason for its causing illness. On chemical examination no mineral poisons were found in samples of the brawn. The author suggested the formation of acrid fatty acids.

Cases of meat poisoning (*Sepsis intestinalis*, according to

Bollinger; infectious enteritis, according to Gaffky) occupied the attention of the medical world for several decades.

Bollinger (1876–80) stressed the importance of meat poisoning in human hygiene. He collected the literature on the subject and drew attention to the relationship between meat-poisoning outbreaks and the septic pyaemic and gastro-intestinal conditions in the animals from which the meat was derived, and also the heat-resisting properties of the poisons associated with such diseases, which were undestroyed by cooking. He enumerated the following diseases of animals, the meat from which, if eaten, might be dangerous to man: septic arthritis of calves after umbilical infection; osteomyelitis and septic wounds or injuries; haemorrhagic enteritis in cattle or calves; septic metritis and septic mastitis in cows; septic peritonitis, pleurisy, and pericarditis. Later, in an address before a medical society in Munich, Bollinger stated that the above assertions had been confirmed, for since that time 11 extensive outbreaks of meat poisoning with about 1,600 cases had been observed, the great proportion of which was of a septic or pyaemic nature.

Gerlach's observations (Ostertag, 1907) upon the connection between the diseases of food animals and cases of meat poisoning are interesting. A cow sustained a severe injury to the udder from a scythe. The wound turned gangrenous and 2 days later the animal was slaughtered. Although Gerlach forbade the consumption of the meat, a portion was consumed by the herder and his family. All were affected with general illness—vomiting, diarrhoea, and extensive weakness.

In a further outbreak, meat from a cow which had been sick after parturition and which was emergency-slaughtered 36 hours later, was eaten by a number of persons. Forty-six became ill and 1 died. The district physician, who did not believe there was any connection between the outbreak and the consumption of the meat, ate some to prove the accuracy of his view; he became dangerously ill.

Klein (1880) carried out some bacteriological examinations in connection with an outbreak of food poisoning (infected ham) at Welbeck, Notts. There was, however, no definite proof that the bacteria isolated caused the illness.

Probably the first bacteriological investigation into the etiology of meat poisoning was made by Johne (1884) in connection with an outbreak which occurred at Lauterbach. A number of persons were affected and 3 died. The animal (a cow) from which the meat

was derived, suffered from enteritis. Johne isolated a bacillus which was pathogenic to mice and other animals and possessed morphological characters similar to those of bacillus anthrax.

Rosenbach (1884) studied and carried out experimental research with staphylococci. He divided them into two species, *Staphylococcus pyogenes aureus* and *Staphylococcus pyogenes albus*, and was able to obtain pure cultures of each on solid media.

Salmon and Theobald Smith, in 1885–6, discovered the American hog organism *B. cholerae-suis*, afterwards named *B. suipestifer* in 1896 by Kruse and later *B. cholerae-suis* by Weldin (1929). The bacillus was apparently not connected at this period with any disease in man.

In May 1888 Gaertner of Jena recorded an outbreak of meat poisoning which occurred at Frankenhausen, caused by the consumption of meat from a cow emergency-slaughtered on account of persistent diarrhoea (enteritis). The appearance of the meat was normal and the organs were not enlarged. There were 59 cases and 1 death: a man who had eaten $1\frac{1}{2}$ lb. of the meat died 36 hours later. Gaertner isolated a bacillus (which he named *B. enteritidis*) from the meat and blood-vessels of the cow and also from the organs of the fatal case. The organism was motile and easily stained. Dogs, cats, chickens, and sparrows were immune, but mice, rabbits, guinea-pigs, and goats were affected when inoculated. The bacillus during growth produced a powerful heat-resisting chemical toxin.

This discovery by Gaertner proved to be a most important landmark in the history of bacterial food poisoning, and *B. enteritidis*, or closely allied forms, have since been isolated during many outbreaks both in this country and abroad.

Johne (1889) demonstrated *B. enteritidis* in the meat from a cow which caused an outbreak of food poisoning at Cotta, Saxony, where 136 persons were affected; 4 died. The meat was eaten raw as well as cooked, thus confirming the findings of Gaertner that the toxin produced by the bacillus was not destroyed by cooking.

One of the most typical and severe outbreaks of meat poisoning caused by *B. enteritidis* (Gaertner) occurred at an industrial girls' school at Limerick, Ireland, in November 1909, and was investigated by McWeeney of Dublin. There were 73 cases with 9 deaths.

No information was available regarding the health of the animal from which the incriminated meat was obtained, except that it could not be fattened. It was killed in a private slaughter-house, and the meat, doubtless of poor quality, sold at a low price.

The general symptoms of the patients were acute gastro-intestinal disturbance accompanied by tenesmus and in some cases collapse.

The meat (stale, but apparently unaltered) was partaken of at noon and the symptoms appeared about 6 p.m. By midnight 28 of the girls were affected. The first death occurred at 7 a.m. the next morning and 8 other children succumbed within the next 2 days. Among the 73 cases every degree of severity was observed, from a condition simulating Asiatic cholera—and which at the autopsy was characterized as 'Cholera Nostras'—to slight headache and malaise with elevation of temperature lasting a few days. There were cases which showed no symptoms at all, but which presented the typical agglutination reaction in the blood and had therefore become infected. From practically all the viscera examined, as well as the discharges from the recovering cases, a typical strain of *B. enteritidis* was isolated. Although the bacillus was very virulent when injected into laboratory animals, guinea-pigs fed with cultures of the organism remained alive. McWeeney failed to infect a dog by feeding it with a large quantity of meat upon which the bacillus had been grown.

McWeeney remarks:

> This severe outbreak of meat poisoning was caused partly by intoxication (cf. the short incubation period), and partly by infection (cf. cultivation of the organism from the 3 fatal and 2 of the recovering cases). The causal micro-organism was the genuine *B. enteritidis* of Gaertner, which must have been conveyed to the sufferers in the beef, and from the history it seems probable that the calf was sickly, and already harboured the bacillus at the time of slaughter.

Ballard (1890) compiled for the Local Government Board an important summary on the then known etiological facts in relation to food poisoning. After intensive research he was able to distinguish between the toxin and the infective form of food poisoning outbreak. Regarding precautionary measures, Ballard wrote:

> What does all this indicate as an efficient precaution against food poisoning? The grand precaution of all is the very common-place one signified by the word 'cleanliness' and it should be the business of all the conservators of Public Health to see that this is observed as well as the business of every master or mistress of a family.

Basenau (1893) isolated *B. morbificans bovis* from the muscles and organs of a cow emergency-slaughtered on account of puerperal fever. On two subsequent occasions he isolated bacilli closely allied to this organism from animals suffering from septic disease.

In America Theobald Smith (1893) investigated the fermentation properties of *B. suipestifer* on different forms of sugar, and his researches established the salmonella group of organisms.

In 1896 Achard and Bensaude isolated an organism to which they gave the name *Bacille paratyphique*. This organism, according to Boycott (1911), was *Salmonella schottmulleri* (*B. paratyphosus B.*).

Durham (England) and de Nobele (Belgium), working independently in 1898, described a bacillus which they had isolated from patients suffering from meat poisoning. This bacillus, which was closely related to *B. enteritidis*, they designated *B. aertrycke* after the name of the Belgian village where the outbreak occurred. The discovery proved to be of paramount importance, as *B. aertrycke* has proved to be the causal organism in a very large number of cases of food infection in this country.

B. aertrycke is now designated *B. typhi-murium*, this being the name given to an organism isolated by Loeffler (1892) from a mouse epizootic and found to be identical with *B. aertrycke*. It is frequently referred to in German literature as the Breslau bacillus, Von Kaensche (1896), and also known as *B. pestis caviae* Wherry (1908), and was classified by Castellani and Chalmers (1919). Its occurrence is widespread throughout the animal kingdom. According to Bergey (1939) it is a natural pathogen for guinea pigs, sheep, parrots, turkeys, canaries, chickens, ducks, and pigeons and has been responsible for numerous food-poisoning outbreaks. In fact, it is one of the most common food-poisoning organisms found in group gastro-enteritis outbreaks, and has been described as found in foods such as meats and duck eggs.

At this time (1892) it was definitely recognized that the *B. enteritidis* type was serologically distinct from other strains as *B. suipestifer* and *B. paratyphosus B.*

Savage (1913) remarks:

> We owe an important advance in the bacteriological study of food infections to Durham, who demonstrated in 1898 that by the use of the agglutination tests the bacilli isolated from food-poisoning outbreaks, hitherto all indistinguishable, could be separated into at least two distinct groups. He also drew attention to the diagnostic value of the examination of the sera of patients suffering from food poisoning.

The work of various observers, including Bainbridge and Boycott, afterwards placed the differentiation and classification on a more sound basis.

Schottmüller (1900) showed that two distinct types of paratyphoid bacilli existed; these were afterwards named *B. paratyphosus* 'A' and 'B' respectively.

Savage (1909) concluded that *B. aertrycke* and *B. para typhosus* 'B' were serologically distinct, and in 1910 Bainbridge and O'Brien carried out agglutination and absorption tests and came to the conclusion that *B. suipestifer* and *B. paratyphosus* 'B' were separate organisms and that *B. aertrycke* strains were identical with *B. suipestifer*, but in 1912 it was recognized that these two strains were not clearly differentiated.

In the Local Government Board Medical Officer's reports from 1906 to 1910 Savage reported on the following important subjects:

(1) The distribution of the Gaertner group in the animal intestine.

(2) The Gaertner group of bacilli in prepared meats and allied foods.

During the period 1909–23, the salmonella group of organisms (designated 'Salmonella' by Lignières in honour of Dr. Salmon who discovered the hog cholera bacillus), a sub-group of the typho-coli group, received a good deal of attention by various observers, both as regards their relationship to one another and their significance in certain illnesses caused by infected food. About this time considerable confusion existed in Europe and America as to which organisms comprised the salmonella group. Much valuable research on the differentiation and classification of the various strains was carried out by Bainbridge (1909–11), Bruce White (1925–6), Boycott (1906), O'Brien (1911), and Savage (1925).

In America a vast amount of important classificatory work on serological lines was carried out by Jordan (1917), Krumwiede (1918), Kohn (1918), and Valentine (1918).

Schütze (1915–20) by means of absorption tests demonstrated the existence of two serological 'aertrycke' types. These were so-called 'Mutton' and 'Newport' types. In 1920 Schütze published an important and advanced work on the subject which recognized a paratyphoid B group, constituted of nine serological types: Schottmüller (*B. paratyphosus* 'B' ipse), Mutton, Newport, Stanley, Binns, Arkansas, 'G', Reading, and Hirschfeld.

Hecht-Johansen (1923) published the results of his extensive study on the classification of the typhoid-paratyphoid group of bacilli.

In 1911 McWeeney published his articles on the etiology of meat poisoning. In the next year Bainbridge, in the Milroy Lectures, gave a detailed review of the whole subject, and drew

attention to the importance of the rat in relation to meat poisoning, and the possibility of the infection of food by these rodents.

In a report to the Local Government Board in 1913 Savage gave a summary of the existing knowledge of bacterial food poisoning and food infections, and in 1920 the Cambridge University Press published his valuable work, *Food Poisoning and Food Infections*. It included a list of British food-poisoning outbreaks from 1878 to 1918.

During the First World War, although enormous quantities of preserved foods were consumed by the troops at home and abroad, only one outbreak of food poisoning was recorded. This was in 1918 at a base in France and was investigated by Perry and Tidy (1919). Over 1,000 men were affected. The epidemic was caused by *B. aertrycke* and was ascribed to a human carrier.

The Ministry of Health issued:

1. In 1921, Memo. 39, Foods on the procedure to be taken for the investigation of outbreaks of illness suspected to be due to food poisoning.

2. In 1935, Memo. 188/Med., to Medical Officers of Health (outside London). In 1949, this Memo. was revised by the Ministry of Health.

A copy of the 1958 revision of the latter publication is appended at the end of this work.

Among the principal works published in America on food infections and intoxications are those by Jordan (1917–31), Damon (1928), Tanner (1933–53), and Dack (1943–9). A large number of important articles on the subject appeared from time to time in American medical publications and scientific literature.

In England, during 1925, two special reports by Savage and Bruce White were issued by the Medical Research Council on *An Investigation of the Salmonella Group, with Special Reference to Food Poisoning*, and *Food Poisoning, a Study of* 100 *Recent Outbreaks*. These were followed, in 1926, by *Further Studies of the Salmonella Group*, by Bruce White. The publication of these very important and comprehensive studies was another landmark in the history of food poisoning, adding as they did materially to the knowledge on the subject. The reports dealt with the identification and classification of the organisms of the salmonella group and the physiological effects produced in animals by the results of salmonella infections: they also described the results of detailed investigations, epidemiological and bacteriological, of 100 actual outbreaks of food poisoning in this country.

Bruce White describes the genus *Salmonella* as a large genus of serologically related, gram-negative and non-sporing bacilli, usual dimensions, but occasionally forming short filaments, showing with certain exceptions a motile peritrichous phase in which they normally occur; in fact, adhering to the pattern of *B. typhosus* in staining properties and morphology. Failing to ferment lactose and saccharose, to clot milk, to liquefy gelatin, or to produce indol, they regularly attack glucose with, but occasionally without gas production. All the known species are pathogenic for man, animals, or both.

During 1926 a Joint Committee of the Royal Sanitary Institute, London, and the Society of the Medical Officers of Health issued a report on clean food.

Savage (1932) delivered the Sedgwick Memorial Lecture in America on 'Some Problems of Salmonella Food Poisoning'.

In February 1940 he opened an important discussion on 'Salmonella Infections' before the Royal Society of Medicine, London, in which he said:

> We have still a long way to go before we can effectively prevent the pathological manifestations of the Salmonella group in man and animals. A potent weapon is an accurate knowledge of the distribution in nature of the various types and of their specialised pathological activities.

A new English translation (edited by Dunlop Young) of an up-to-date edition of Ostertag's *Meat Inspection* was published in 1934. The history of meat poisoning in Germany is given in detail.

Smith (1934) described *Salm. aberdeen*. This organism has seldom been isolated from cases of food poisoning, but Brockbank, Metcalfe Brown, and Parker (1950) recorded a widespread outbreak following the consumption of meat pies infected with *Salm. aberdeen*. Up to this time there had been nothing to suggest that this organism was a potential danger to man.

Jordan (1934) isolated *Salm. panamà* during an outbreak among American troops in the Panama Canal zone.

Hormaeche and Peluffo (1936) in Uruguay reported the isolation of *Salm. montevideo* from a monkey and other animals. Bruner and Edwards (1940) in Minnesota described *Salm. meleagridis*. The above organisms have now a wide geographical distribution.

In November 1940 the Ministry of Health issued an important memorandum (Cir. 2198; 25.11.40) to sanitary authorities on the subject of 'Precautions against the Spread of Alimentary Infections'. The memorandum reminds local authorities of the

measures which can usefully be taken to protect the public against the spread of the diseases commonly conveyed by food, i.e. diseases of the enteric group (typhoid and paratyphoid fevers), dysentery, food poisoning and intestinal parasitism.

Lerche and Bartel (1943) in the course of five years, 'typed' some 7,674 salmonella strains. They investigated the main pathological conditions caused by the various types and also worked out their geographical and annual distribution. During the above period the increase or decrease of certain types was noticed. They also observed that the *Salm. dublin* type occurred more frequently in cattle than in other animals, although at times this organism was found in pigs, sheep or horses. *Salm. breslau* (*Salm. typhi-murium*) was commonly isolated from cattle. Systematic investigations by these workers revealed that the so-called new types of salmonella are relatively rare in livestock, and a small percentage of those causing human infections are found in animals.

In America, during the past few years, several outbreaks of illness have been caused by the consumption of certain foods—mostly milk products, particularly cream cakes, custards, or puffs, etc., infected by a toxigenic staphylococcus.

The experimental evidence that certain staphylococci produce gastric irritation has been provided mainly by Dack, Jordan, and their colleagues. Jordan in summarizing the information expressed the opinion that probably many outbreaks due to staphylococci have been overlooked. These organisms are widespread in nature and consequently this opens up very considerable possibilities as a cause of obscure outbreaks.

Dolman (Canada) studied the subject and carried out some valuable experimental research. In his writings on bacterial food poisoning (1943), he gives prominence to illness due to extero-toxin-producing staphylococci, and considers it 'the commonest, and in some respects, the least controllable form of food poisoning'.

Among the recent and foremost research investigators of food-poisoning outbreaks should be mentioned Dr. W. M. Scott, who lost his life through enemy action in 1941.

Scott was an acknowledged expert in the field of bacteriology, and devoted much attention to the salmonella and dysentery bacilli, and, in collaboration with others, defined several new salmonella types, or determined the association of types previously found only in animals with human infections. In 1930 he drew attention to the relation of duck eggs with food poisoning

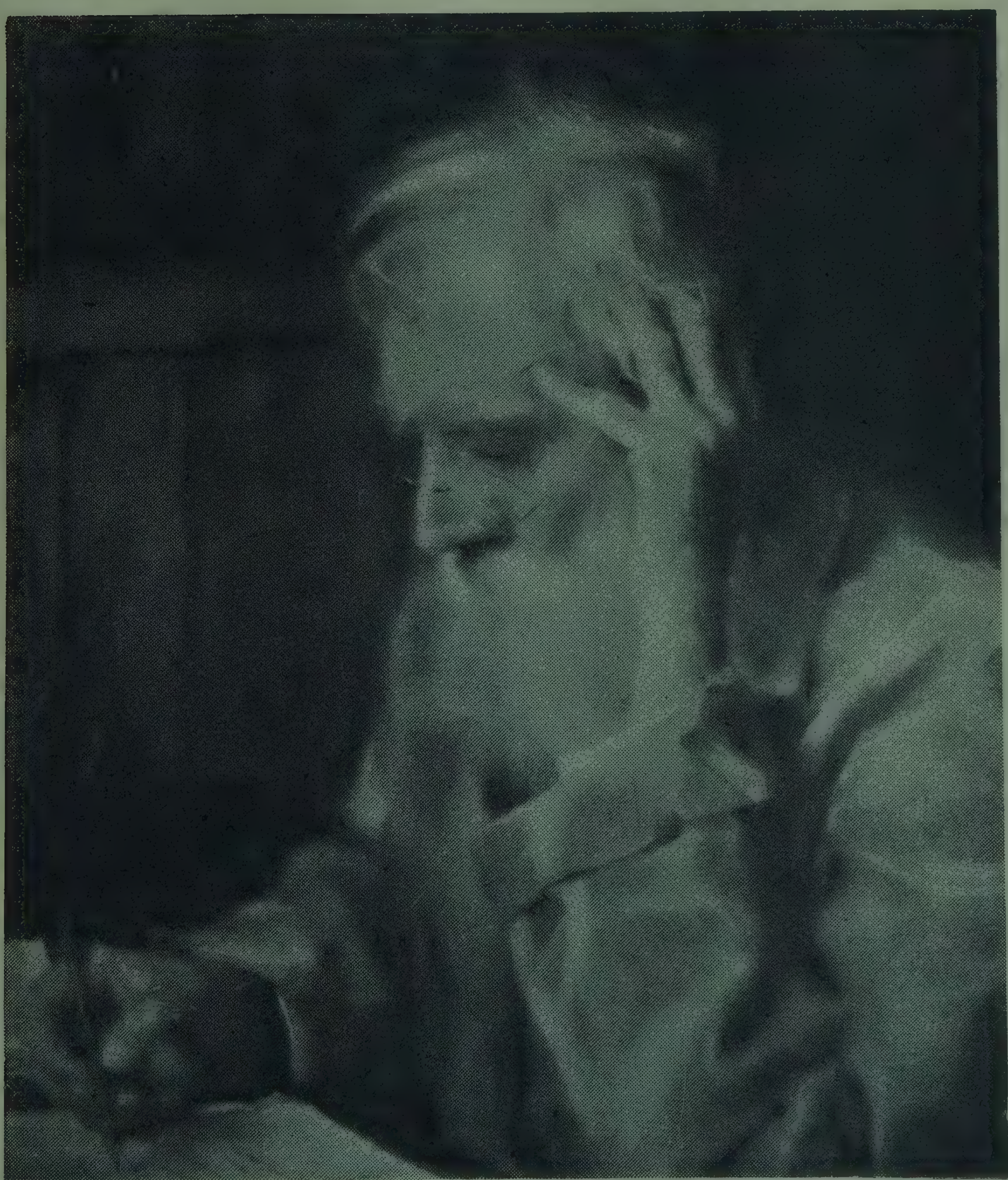
PLATE 1. Sir William G. Savage, M.D.

(*a*) Professor A. Gaertner

(*b*) H. E. Durham, Sc.D., M.B., B.C., F.R.C.S.

PLATE 2

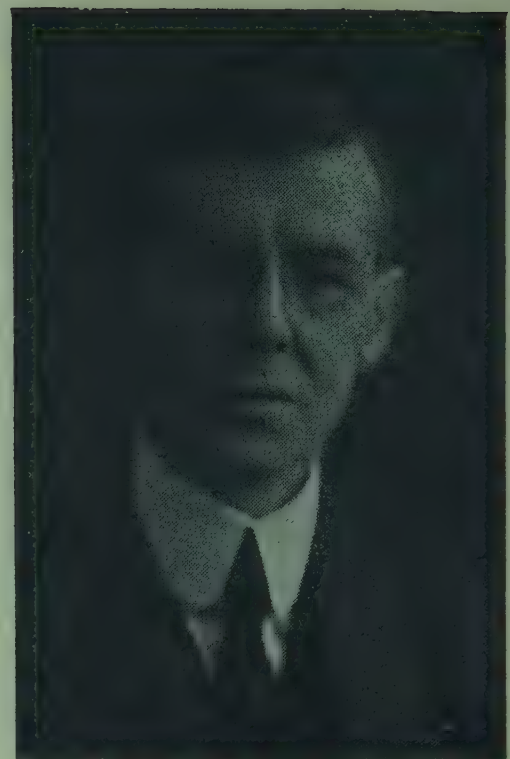
(*a*) Professor E. J. McWeeney



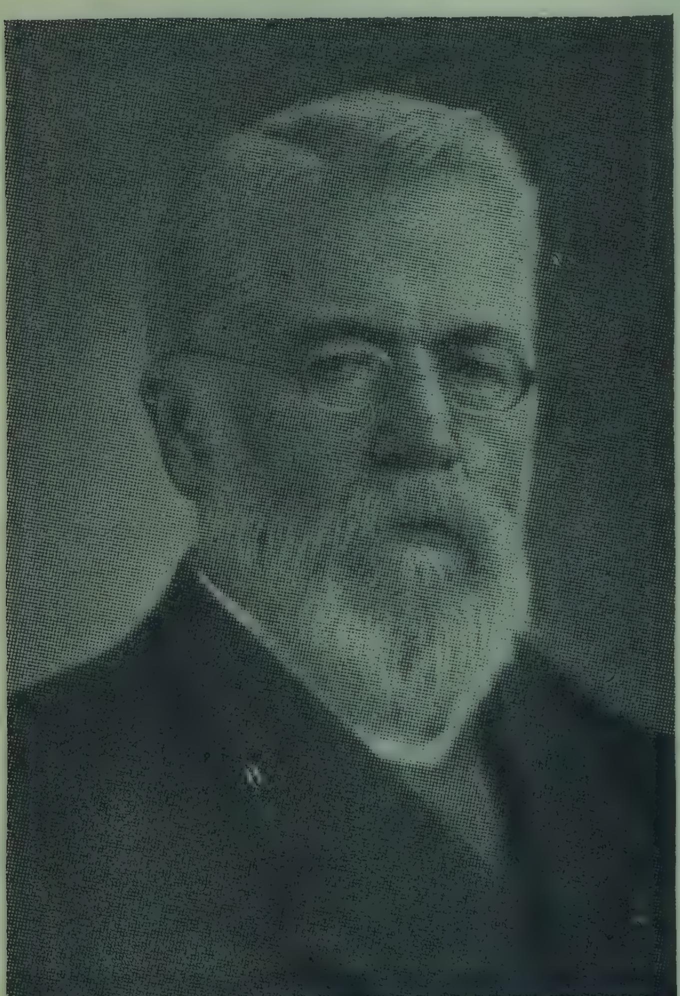
(*b*) Edward Ballard, M.D., F.R.C.P., F.R.S.

PLATE 3

(*a*) Professor Theobald Smith



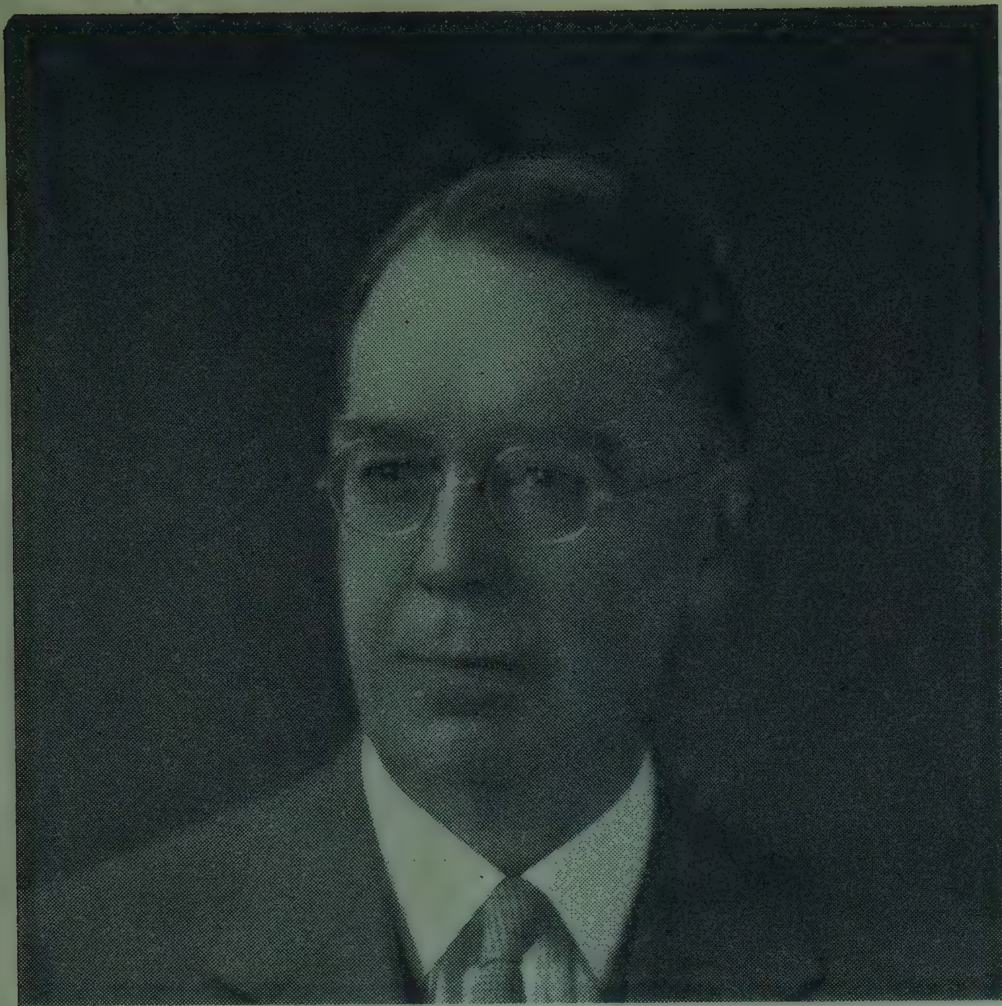
(*b*) Professor F. A. Bainbridge



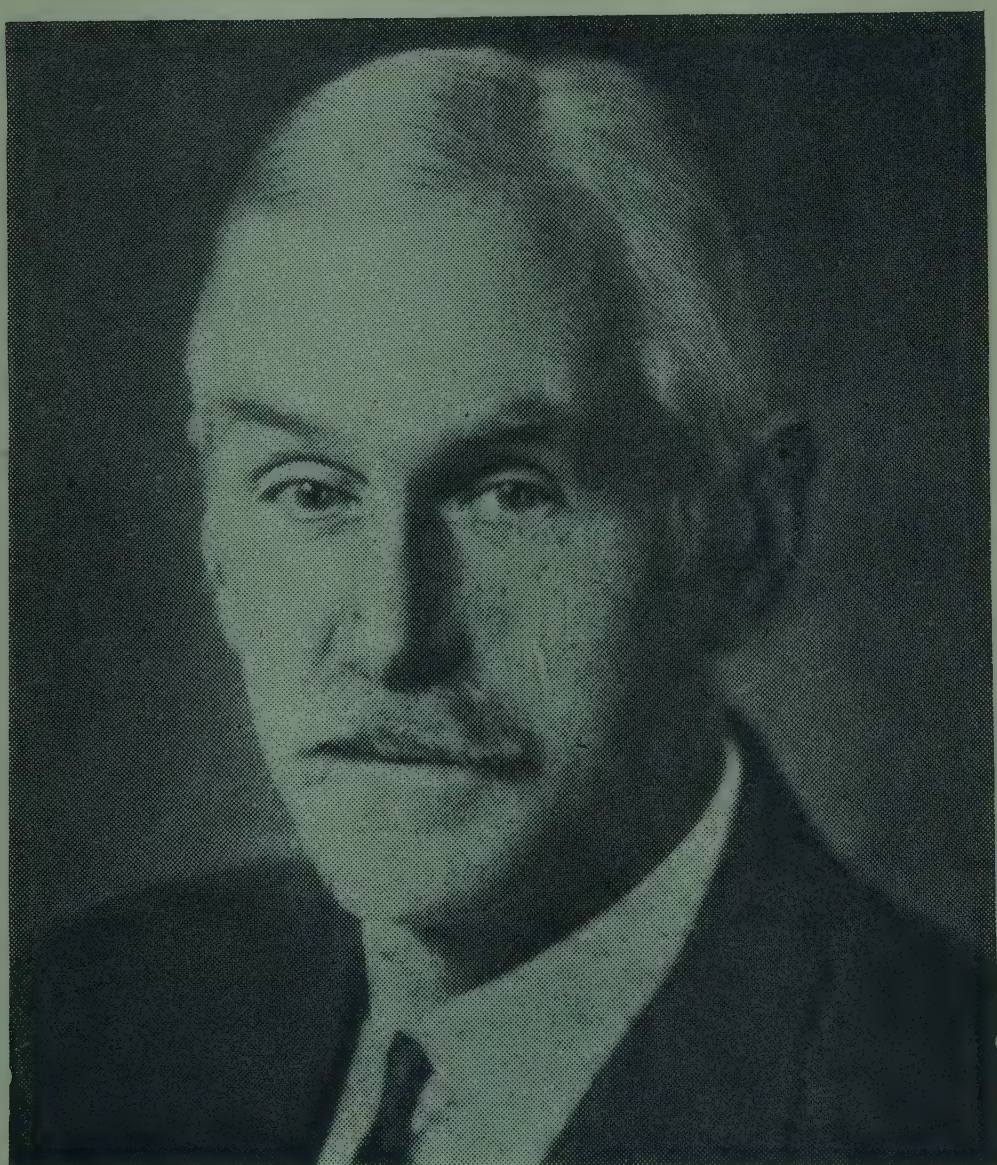
(*c*) Otto von Bollinger

(*d*) W. M. Scott, M.D.

PLATE 4

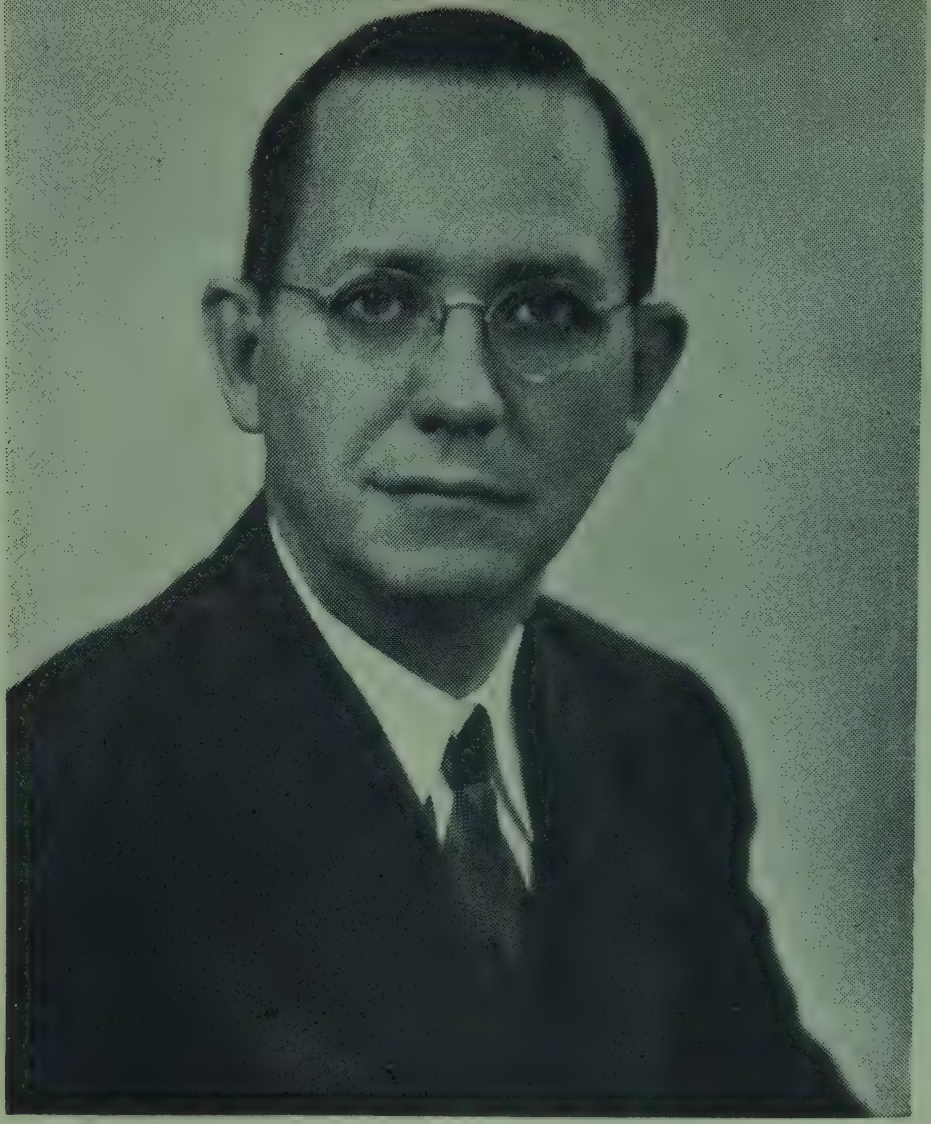


(*a*) Dr. Edwin Oakes Jordan



(*b*) Professor A. E. Boycott

PLATE 5



(*a*) Dr. Gail M. Dack

(*b*) Professor C. E. Dolman, M.B., D.P.H.

PLATE 6

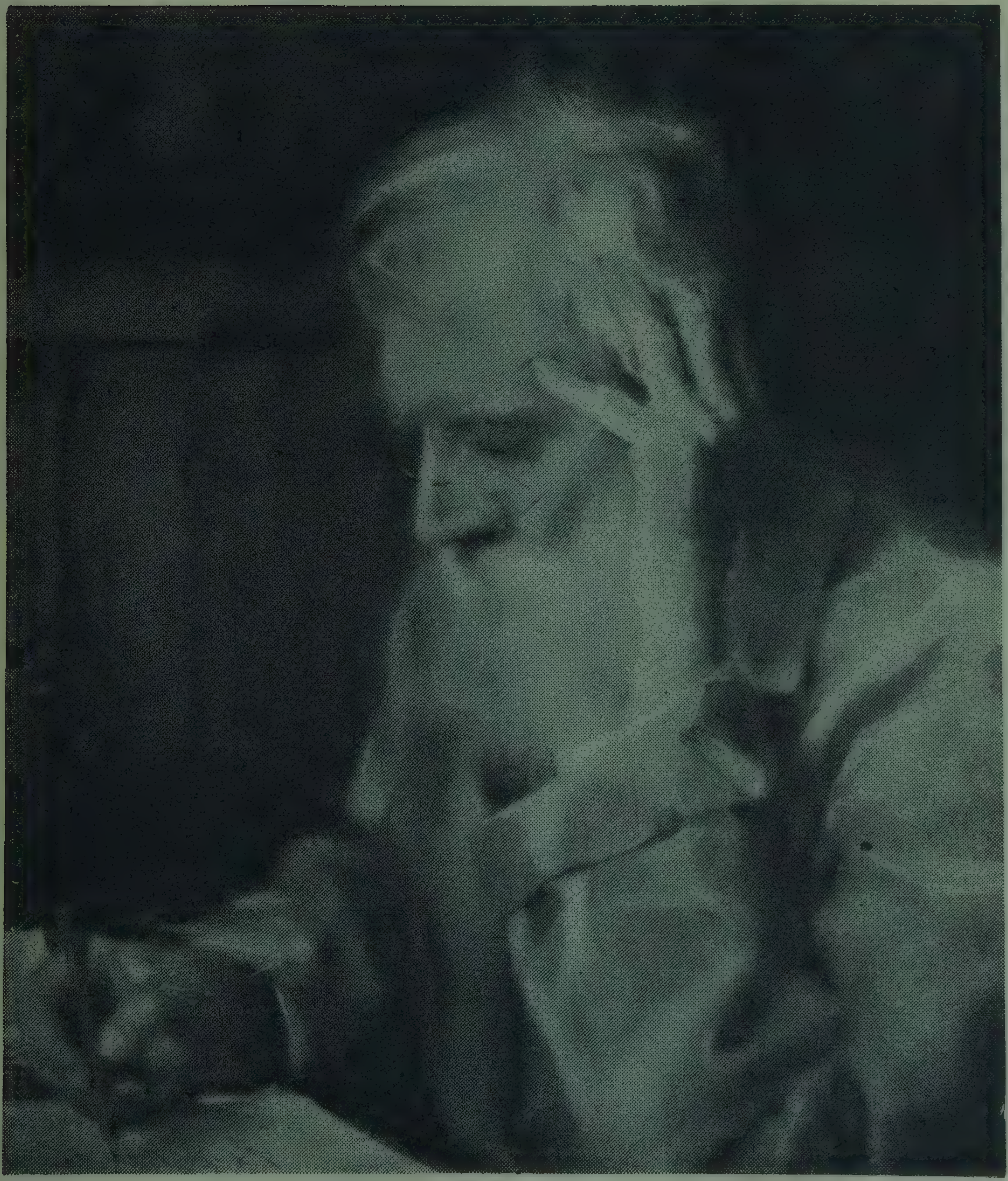
PLATE 1. Sir William G. Savage, M.D.

(*a*) Professor A. Gaertner

(*b*) H. E. Durham, Sc.D., M.B., B.C., F.R.C.S.

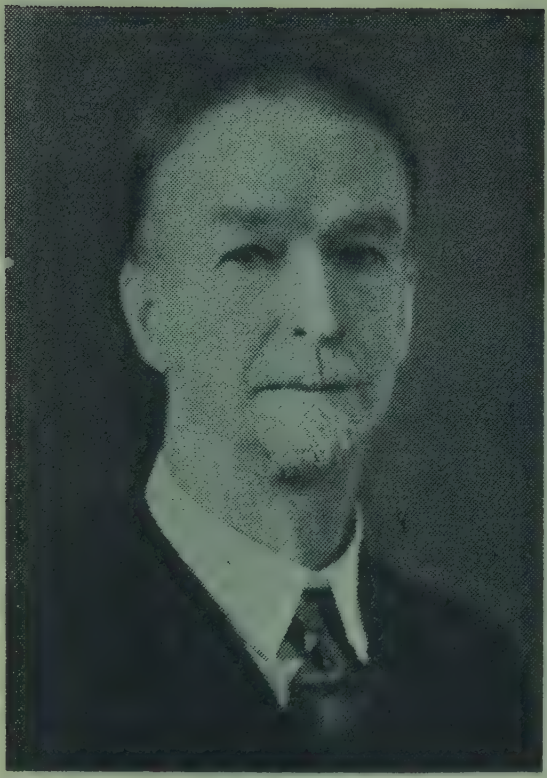
PLATE 2

(*a*) Professor E. J. McWeeney



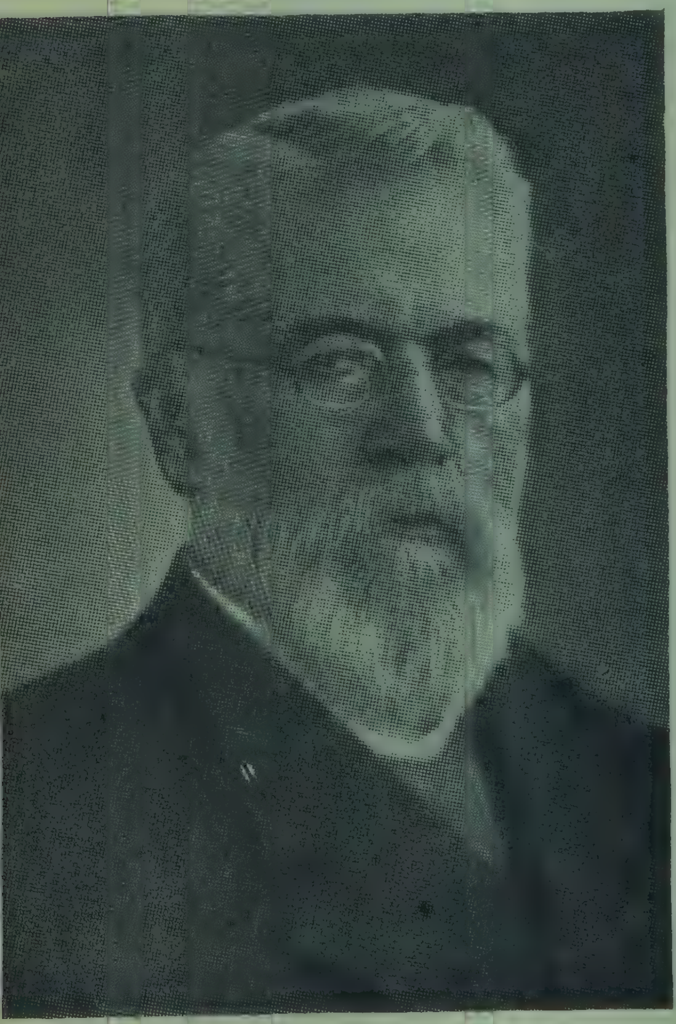
(*b*) Edward Ballard, M.D., F.R.C.P., F.R.S.

PLATE 3

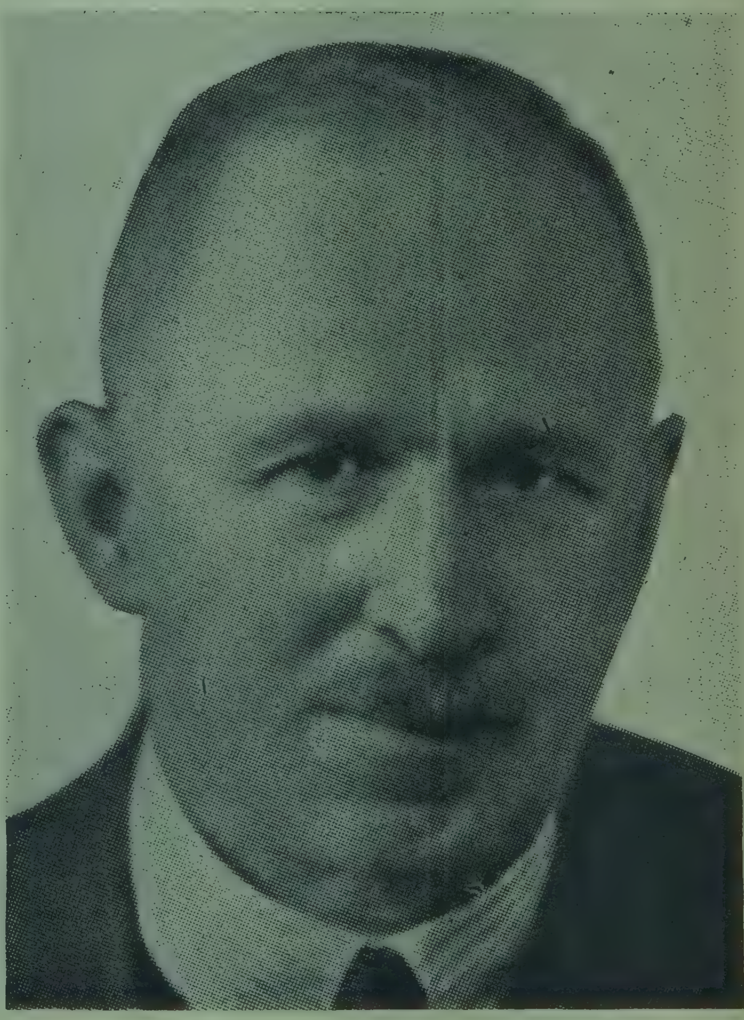


(*a*) Professor Theobald Smith

(*b*) Professor F. A. Bainbridge

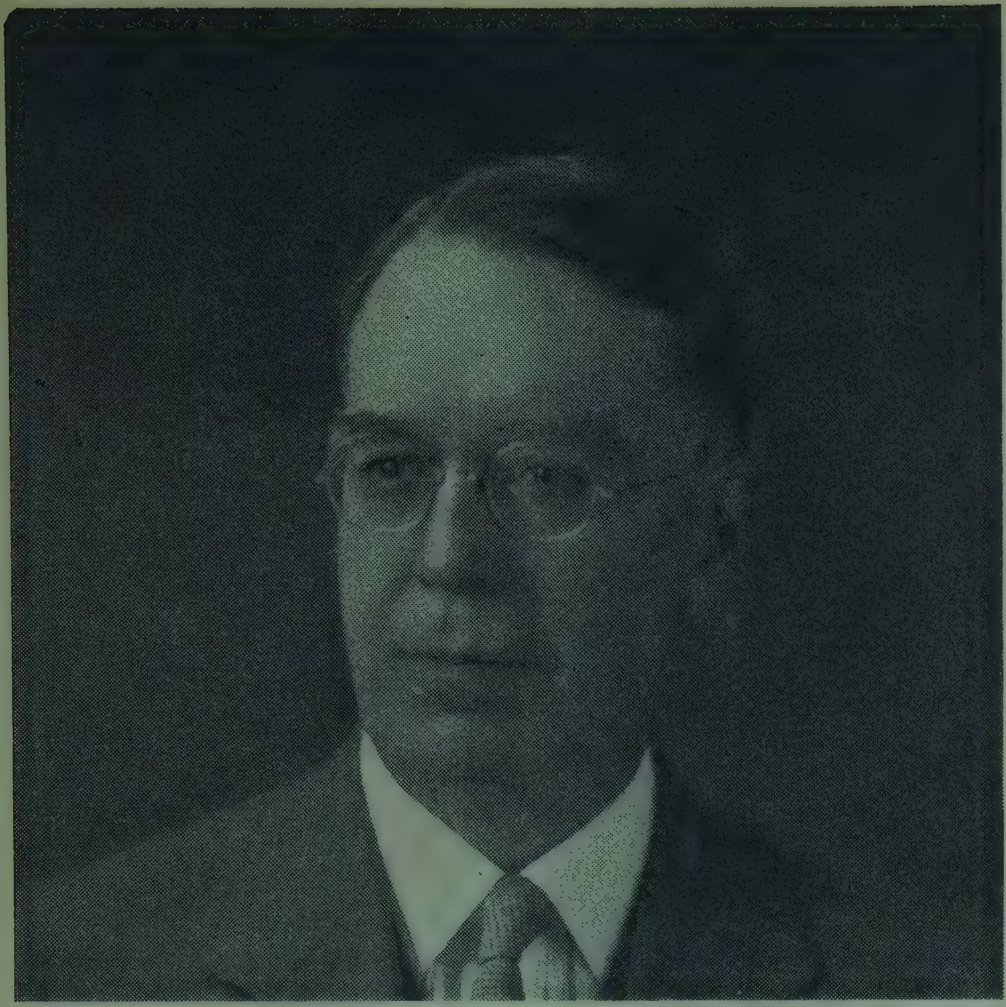


(*c*) Otto von Bollinger

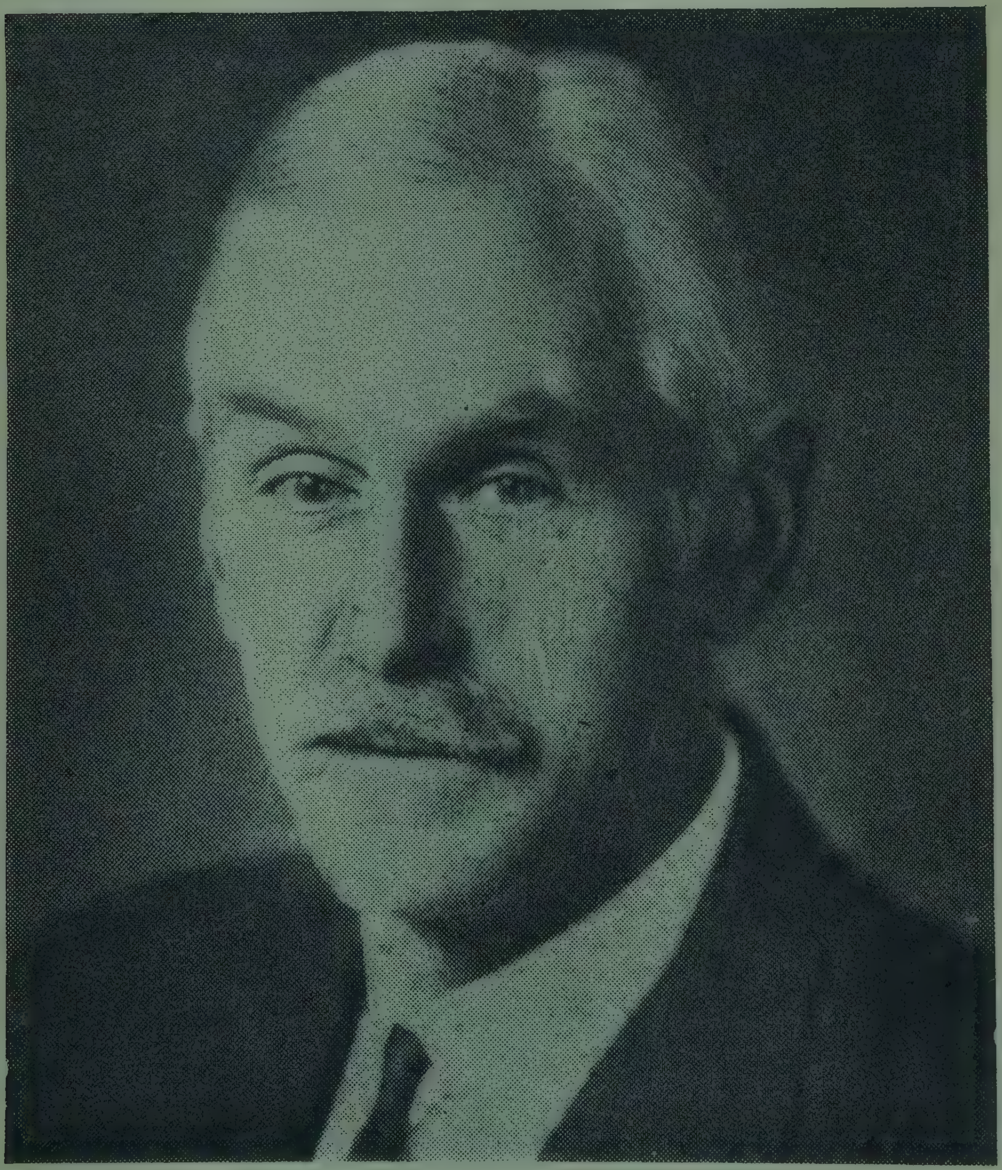


(*d*) W. M. Scott, M.D.

PLATE 4



(*a*) Dr. Edwin Oakes Jordan



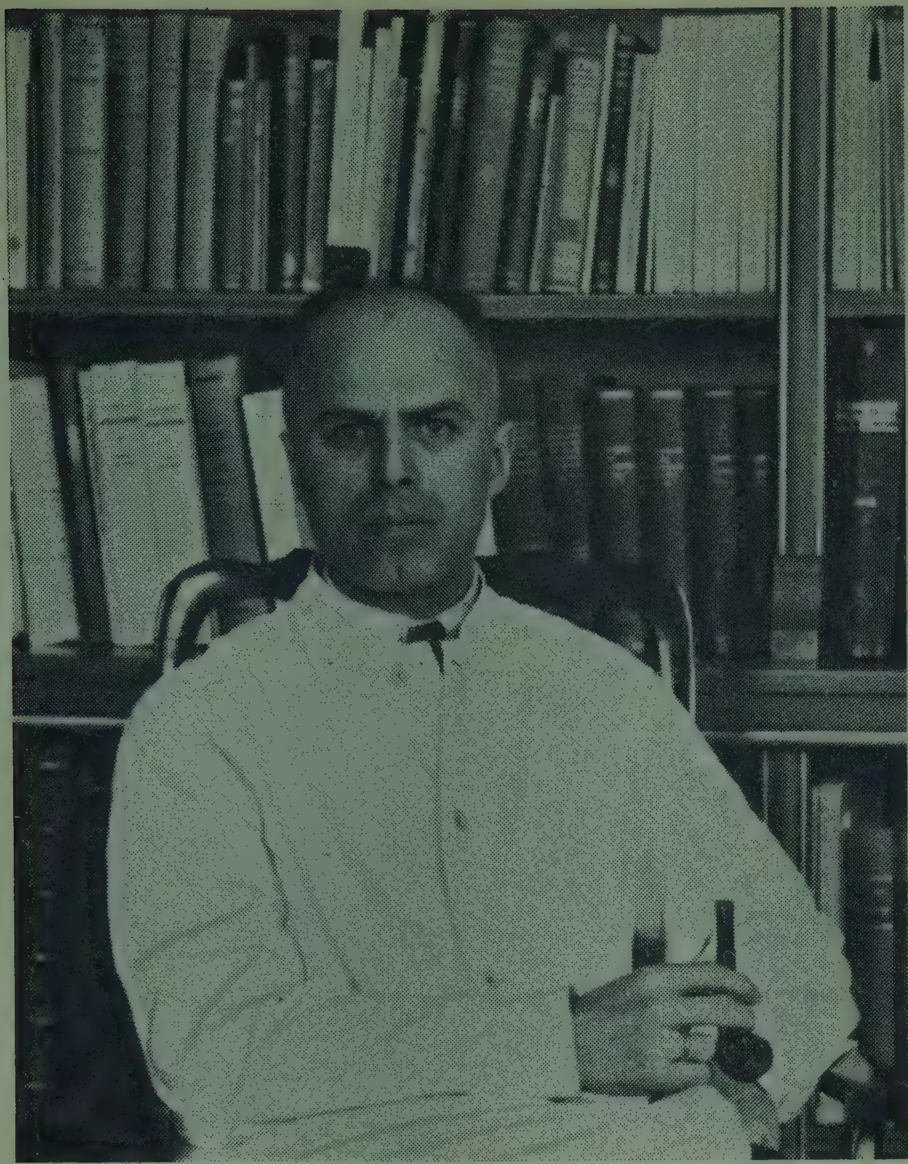
(*b*) Professor A. E. Boycott

Plate 5

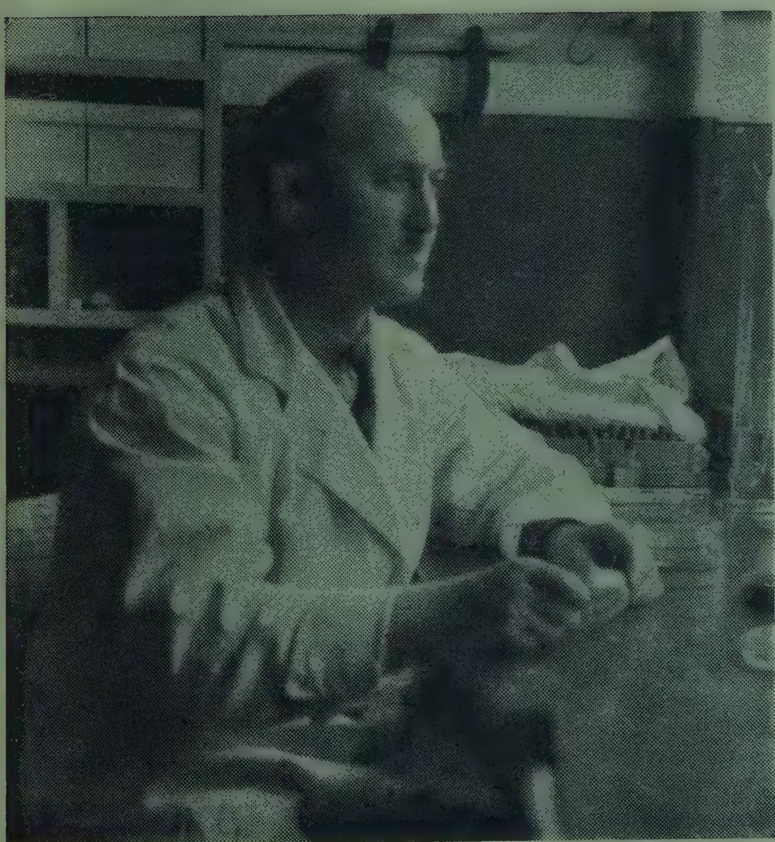
(*a*) Dr. Gail M. Dack

(*b*) Professor C. E. Dolman, M.B., D.P.H.

PLATE 6



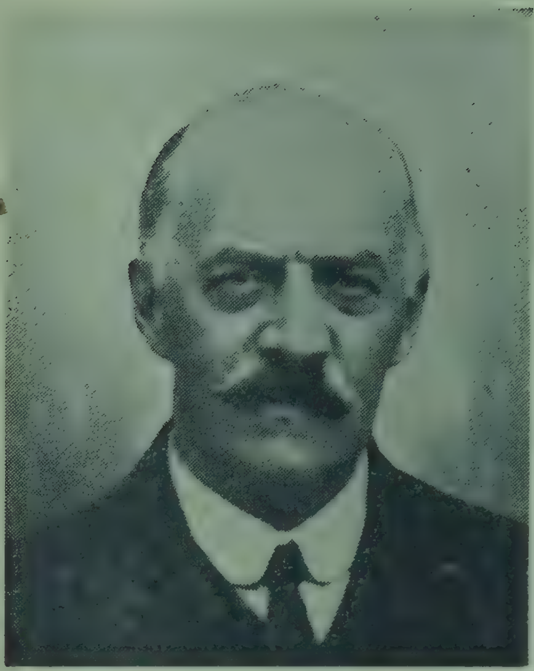
(*a*) Dr. F. Kauffmann



(*b*) P. Bruce White, B.Sc., F.R.S.

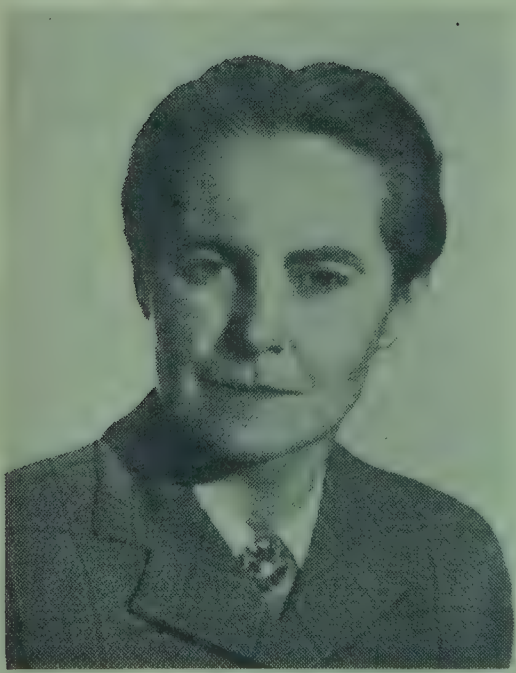
(*c*) Professor F. Wilbur Tanner

PLATE 7



(*a*) Dr. G. S. Graham Smith

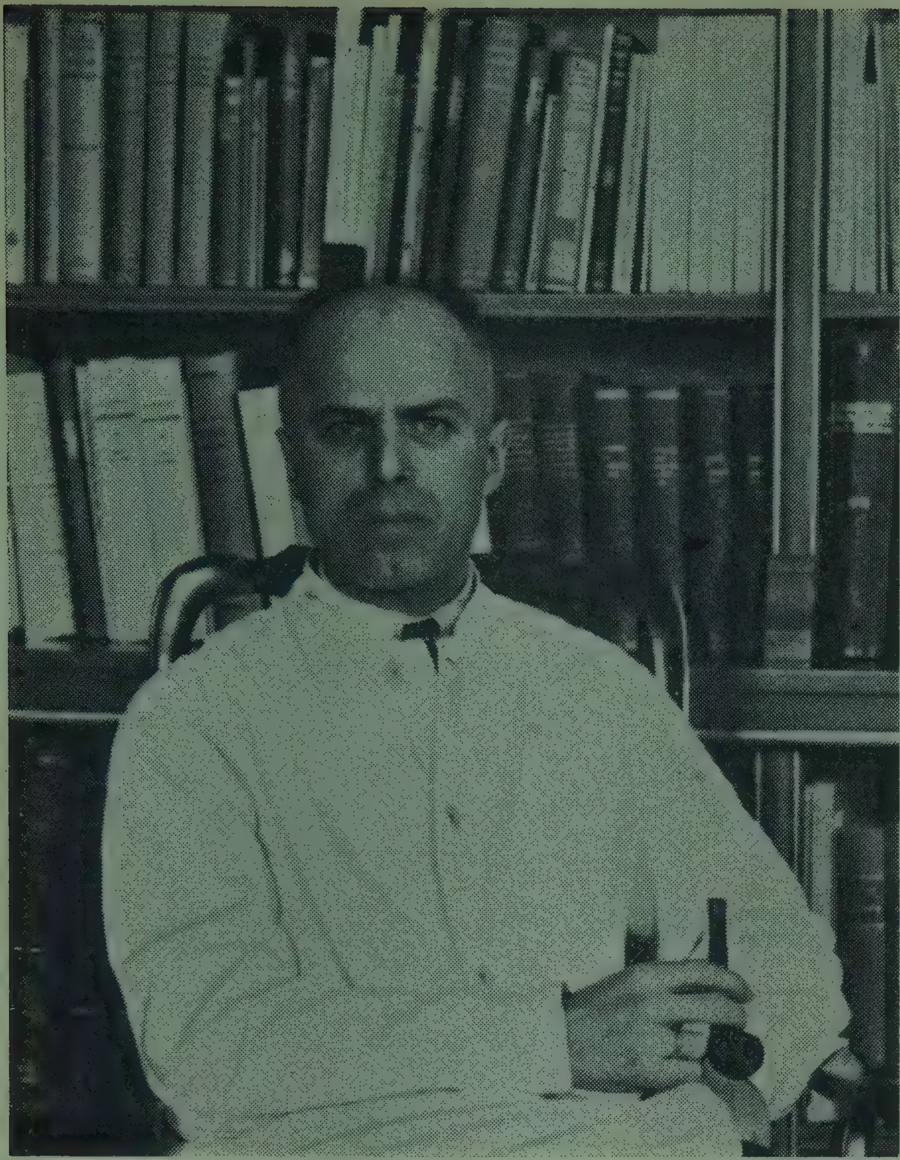
(*b*) Sir G. S. Buchanan



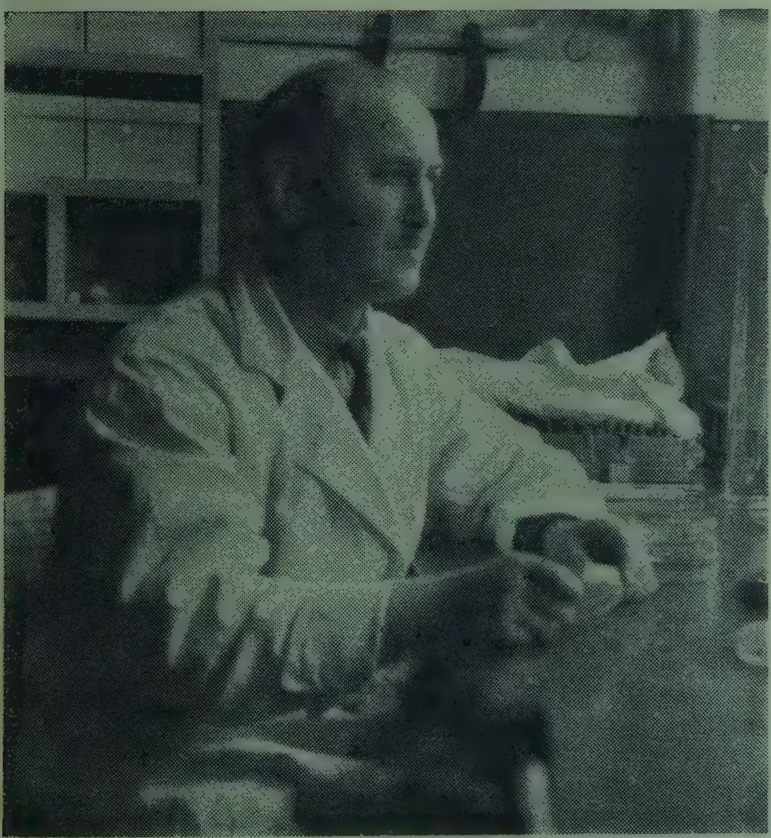
(*c*) Betty Hobbs,
D.Sc., Ph.D.

(*d*) Dr. Joan Taylor,
B.Sc., M.B., M.R.C.S., L.R.C.P.

PLATE 8



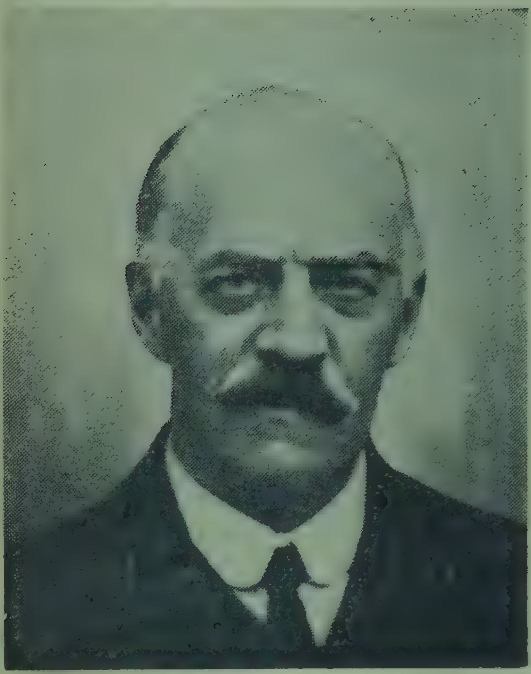
(*a*) Dr. F. Kauffmann



(*b*) P. Bruce White, B.Sc., F.R.S.

(*c*) Professor F. Wilbur Tanner

PLATE 7



(*a*) Dr. G. S. Graham Smith

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(*c*) Betty Hobbs,
D.Sc., Ph.D.

(*d*) Dr. Joan Taylor,
B.Sc., M.B., M.R.C.S., L.R.C.P.

PLATE 8

in man, and later was able to prove, through the isolation of *Salm. typhi-murium* from duck eggs and from the ducks of three flocks concerned with outbreaks of food poisoning, that eggs were the vehicle of infection.

The subject of food infection and intoxication has received much attention in the annual reports (On the State of the Public Health) of the Chief Medical Officer of the Ministry of Health. The numbers and particulars of the various outbreaks that have occurred during each year are given, together with other interesting and valuable information and advice.

The Ministries of Health and Food (1946) issued a press warning 'Never to eat raw sausage meat' and advised the public not to eat pork, pork-sausage meat, ham or any pork products unless it has been thoroughly cooked.

During 1946 the Public Health Laboratory Service was created by the Ministry of Health. This service maintains in England and Wales 8 regional, 48 area, and 2 associated laboratories. There are also 17 others recognized only for the purpose of sanitary bacteriology. To the above, specimens etc., obtained during the investigation of outbreaks of food poisoning can be forwarded for bacteriological examination or chemical analysis.

In the Headquarters Establishment at Colindale, London, there are 10 laboratories, including the Food Hygiene Laboratory, the Salmonella Reference Laboratory, the Streptococcus and Staphylococcus Reference Laboratories, and the Epidemiological Research Laboratory.

In 1947 a Special Report Series No. 260 was issued by the Medical Research Council, London, on the bacteriology of spray-dried egg with particular reference to food poisoning. The report contains a record of the bacteriological and epidemiological investigations carried out on imported spray-dried egg of American origin.

In 1948, a Catering Trade Working Party was appointed to make recommendations to the Ministries of Food and Health and the Secretary of State for Scotland as to the precautions considered practicable and desirable with a view to securing the observance of sanitary and cleanly conditions in the catering trade. The recommendations were embodied in a report published in 1951.

A working party was also appointed in 1949, to review present trade practice and legal requirements for securing that conditions in the meat manufacturing trades are clean and sanitary and that products, and the materials from which they are prepared, are

wholesome in all respects; to consider whether new or amended requirements by way of statute or regulation were desirable; and to make recommendations to the Ministries of Food and Health and the Secretary of State for Scotland. The Report by the working party was published in 1950.

The Minister of Food, in 1949, issued Model Byelaws (Series I) dealing with the handling, wrapping, and delivery of food and the sale of food in the open air.

In 1950, F. Kauffmann, Chief International Salmonella Centre, State Serum Institute, Copenhagen, compiled a monograph on the diagnosis of salmonella types and in the following year published his collected studies on Enterodacteriaceae.

The Ministry of Health in 1953 circulated the new Public Health (Infectious Diseases) Regulations. Important provisions were made in these regarding the action to be taken by local authorities and medical officers of health for the prevention of food poisoning.

In the same year the Ministry of Health circulated a memorandum with appendices on the subject of hygiene in hospital catering departments, reminding hospital authorities of the dangers of food poisoning. The memorandum suggested that in each hospital a medical officer should be responsible for advising the hospital authorities and the catering officer on hygiene and for arranging lectures to be given on the subject to the catering staff.

In 1954, the Minister of Education issued a Circular (No. 272) to all local educational authorities on 'The Prevention of Food Poisoning in School Canteens'.

During 1955, a new Food and Drugs Act came into operation. Part V (Administration) of the Act provides for the constitution of a Food Hygiene Advisory Council, to which the Ministers can refer from time to time for consideration and advice questions relating to this Act as it applies to food.

In December 1955 the Minister of Agriculture, Fisheries and Food, and the Minister of Health, in exercise of their powers conferred on them by certain sections of the above Act, made and issued the important Food Hygiene Regulations, which came into operation on 1 January 1956. These add new provisions in respect of the hygienic handling of food and the construction and maintenance of premises, stalls, vehicles, etc., where food is handled. They also replace certain regulations of the Public Health (Meat) Regulations, 1924, in respect of the transport and carrying of meat.

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Chapter III

BACTERIAL FOOD POISONING

THIS type of food poisoning signifies illness due to the ingestion of some particular article of food which either contains living pathogenic organisms capable of setting up acute inflammation of the alimentary tract, i.e. 'food infection' or irritative poisonous substances (toxins) only, which have been produced during the growth and multiplication of the pathogenic organisms in the food prior to ingestion, i.e. 'food intoxication'. These poisonous substances retain their potency even after exposure to temperatures sufficiently high to destroy the bacteria producing them.

It has been calculated that approximately 40 per cent of outbreaks of food poisoning are of the infection type, 20 per cent of the toxin type, and 40 per cent of the non-specific type.

THE SALMONELLA GROUP

The numerous researches and investigations into outbreaks of food poisoning, together with the study of the organisms isolated, by Bainbridge, Boycott, Durham, Savage, Bruce White, Scott, Wilson, Taylor, Hobbs, and others in this country: by Jordan and his colleagues, Geiger, Meyer, Tanner, Dack and many other observers in the United States of America, and by Kauffmann in Germany, demonstrated that certain recognized types of the salmonella group are a cause of food poisoning. Moreover, the valuable and intensive research work of Bruce White (1926, 1929, 1930, 1931, and 1932), Kauffmann (1929, 1930, 1931, 1934, 1939, 1941 and 1948), and Edwards and Bruner (1942), provided a reliable scheme for the classification and differentiation of this group of organisms.

The Kauffmann-White classification was recommended for adoption by the Salmonella Sub-committee of the International Society of Microbiologists, 1934, and an international salmonella centre was established at the Statens Seruminstitut, Copenhagen. Owing to the large extension of this group, Kauffmann (1947) rearranged the diagnostic antigenic scheme to make it more surveyable and as simple as possible; 142 types were included in the scheme. In recent years researches and bacteriological examinations, carried out at home and abroad in connection with

food-poisoning outbreaks, have revealed a very large number of new types in the salmonella group which have been separated and distinguished by their antigenic differences and classified by bacteriophage typing. Nearly 400 distinct serotypes have now been differentiated.

The salmonella organisms which are primarily intestinal parasites and widely distributed in man, mammals and birds (emphasizing the general lack of host specificity) are all closely related to each other, but can be distinguished by cultural or serological tests. Henning and Haig (1939) published an extensive paper on the antigenic structure of the salmonellas obtained from domestic animals and birds. Edwards and Bruner (1943) recorded their investigations into the occurrence and distribution of salmonella types and the serological recognition of members of this group has been described by Bridges and Taylor (1944).

The members of the salmonella group are short (being about 2–3μ long and 0·5–0·7μ broad) motile (by peritrichate flagella), gram negative, non-sporing rods with rounded ends. They ferment glucose, maltose, mannitol, dulcite, and sorbitol with the production of acid and gas. Lactose, sucrose, adonitol, and salicin are not fermented. There is no production of indol or acetylmethylcarbinol, while the formation of H_2S varies with the different types of organisms. Gelatin is seldom liquefied and milk is not coagulated. Salmonella organisms have very little resistance to heat, being killed at 55°C. (131°F.) for 1 hour; or 60°C. (140°F.) for 15–20 min. They grow readily on ordinary culture media, at temperatures ranging from 21° to 36°C. (70°–80°F.). After 2 hours growth at 37·2°C. (99°F) reproduce about every 15 minutes.

Koser (1922) found that all members of the group multiplied rapidly in the liquor of several cooked vegetables, with the exception of highly acid sauerkraut. In fruit juices a rapid destruction of the organisms occurs.

Some of the salmonella types can be isolated from animals which apparently are free from disease, but evidence favours the view that they are only present in the carrier state usually as a result of previous active infection. Savage (1920) wrote:

> These bacilli are not *natural* inhabitants of the animals used for food and not found more frequently than can be accounted for on the supposition that their presence is due to an actual cause of disease or the carrier state after infection.

The possibilities of direct transference of salmonella infection from animals to man are obvious.

Outbreaks due to members of the salmonella group are met with in many parts of the world. They are common in Europe and America, and cases have been reported from the widely scattered areas of Asia and Africa. Poisoning of the gastro-intestinal type is caused usually by organisms of the salmonella group. They multiply in the intestinal tract and give rise to a true infection. Occasionally, however, other organisms are associated with food poisoning, such as *Staphylococcus aureus*, *Cl. welchii*, *Proteus vulgaris*, and members of the dysentery group (*Sh. sonnei* and *Sh. flexneri*).

The salmonella strain most commonly isolated in British food-poisoning outbreaks is *Salm. typhi-murium* (formerly *B. aertrycke*) so named by Loeffler (1892). He isolated the bacillus during a mouse typhoid epidemic in the Hygiene Institute of Greifswald, Germany. It is a cause of disease in several of our food animals and in other animals, rodents and birds. Its reservoir is not man but of late years it seems to be evolving into a human pathogen. Owing to the wide distribution throughout the animal world, this organism is generally considered the most prevalent cause of meat-borne salmonellosis in man.

The gradual increase in food poisoning incidents due to *Salm. typhi-murium*, as compared with other causal organisms is shown in the following table, for the 5 years 1951–5.

Causal organism	1953	1954	1955	1956	1957
Salm. typhi-murium	2,438	3,038	4,276	3,176	2,931
Other salmonellae	709	520	1,070	1,147	1,287
Staphylococci	132	127	138	131	128
Cl. welchii	25	48	90	77	93
Other organisms	6	9	12	16	6
Not discovered	2,000	2,322	3,447	3,163	2,625

(Compiled from Annual Reports on Food Poisoning contained in *Mon. Bull. Minist. Hlth. Lab. Ser.*)

The other types most frequently isolated are *Salm. enteritidis*, *Salm. thompson*, and *Salm. newport*. *Salm. enteritidis*, which is found associated with diseased cattle, produces in man a virulent form of food poisoning. Outbreaks due to this organism, however, are more common in countries other than Great Britain and tend to be more severe. It is frequently isolated from emergency-slaughtered cattle in Germany. Bruce White (1929) differentiated

the *Salm. dublin* type from *Salm. enteritidis* strains, this particular strain coming from a fatal case of continued fever in Dublin. After its differentiation it was possible to show that it was especially associated with calves (calf dysentery) and cattle. Knoth (1936), examining meat from slaughtered animals, found that of 538 strains from calves, 506 were *dublin* types, and 17 out of 18 from adult cattle were also *dublin*. This was confirmed later by several other observers.

Savage (1940), in a discussion on salmonella infections before the Royal Society of Medicine, pointed out that :

> Since the differentiation of dublin it has been found to be the cause of human infections in a number of cases. No doubt a number of outbreaks due to *Salm. enteritidis*, especially from milk, were due to *dublin*, but as the strains have not survived to be differentiated we have no accurate knowledge.

In recent years *Salm. dublin* has been fully recognized as a cause of bovine infections. It infests fewer species, however, than other types of salmonellae, the most common being the cow. An apparently healthy animal may be a carrier of this organism and so induce a widespread outbreak of milk-borne food poisoning.

The rapid multiplication of *Salm. dublin* in milk when stored at room temperature may explain why large epidemics are caused sometimes by a single cow and also why infected milk may often not cause food poisoning if drunk in a fresh condition. The organism causes a serious type of illness in man and sometimes bone infections: see Purnell (1952), Miller (1954).

A milk-borne outbreak due to *Salm. dublin* has been recorded by Sutherland and Berger (1944). The outbreak occurred in the West Riding of Yorkshire in May 1943: 162 cases were notified but no deaths occurred. The investigators state:

> The source of infection was an apparently healthy cow which was excreting large numbers of the organism in the dung. The carrier cow was identified by the agglutinin content of its milk. The milk of normal cows did not possess any agglutinins for S. dublin.

The onset of the illness was sudden. The symptoms, which commenced about 14 to 20 hours after ingestion of the vehicle of infection, were: sickness or actual vomiting, diarrhoea (there was no blood or mucus in the motions), headache, and a febrile period which lasted for about 3 days. In a small number of cases a profuse herpetic eruption appeared on the lips and nose. The relatively long incubation period suggested infection with living salmonella organisms.

The recorders remark:

How the milk became the vehicle of infection is a matter of conjecture. As the dung of the carrier cow contained the organism in large numbers, it seems probable that the milk became contaminated as a result of faulty methods of production at the farm. On the other hand, although the milk of this cow was repeatedly examined with negative results, the possibility of intermittent excretion of S. dublin in the milk cannot be excluded.

Salmonella Types	*Disease-Producing Role*	
	Man	*Animals, etc.*
Salm. typhi-murium	Gastro-enteritis (food poisoning). The commonest (83 %) type of all those isolated.	Cause of enteritis in rats, mice, guinea pigs and other rodents, dogs, cats, birds, (including parrots). Occasionally found in pigs and has been a cause of calf enteritis. Infection in ducks, duck eggs, hens and their eggs.
Salm. enteritidis (including sub-strains)	Gastro-enteritis of food poisoning type, occasionally septicaemia.	Disease in cows and calves. Sometimes found in pigs, ducks, and other animals. Epidemics in rats and mice.
Salm. thompson	Food poisoning.	Chicks, geese, eggs, pigs.
Salm. newport	Food poisoning outbreaks and sporadic cases.	Dogs suffering from enteritis, pigs.
Salm. dublin	Food poisoning.	Widely distributed in cattle and foxes.
Salm. bovis-morbificans	Food poisoning.	Original strain from cows suffering from puerperal metritis.
Salm. cholerae-suis	Food poisoning.	Pigs.
Salm. derby	Food poisoning.	Pigs, exact disease role unknown.
Salm. stanley	Food poisoning.	—
Salm. anatum	Food poisoning.	Ducks, chickens, turkeys, dogs, and cats.
Salm. paratyphi B	Paratyphoid fever, causing long continued infection, but rarely gastro-intestinal symptoms.	Pigs, dogs, cats.

Field (1948) records the results of investigations made during 1946-7 in West Wales, where bovine salmonellosis was diagnosed on 70 farms. Affected animals on 67 farms were over 1 year old and all except one were females. Calves were affected on the two remaining farms. Specimens from affected animals on 66 farms yielded *Salm. dublin* and on 4 farms *Salm. typhi-murium.* The

mortality rate was estimated at approximately 70 per cent. Fifty-three of the outbreaks occurred on farms in the County of Carmarthen. In infected adult cattle an initial fever is accompanied by depression and decreased milk yield. During this phase the organism is present in the blood and milk. This stage is followed within 24 hours by dysentery and the organism can readily be isolated from the faeces. The majority of affected animals die within 1 to 5 days of the onset of symptoms. A few recover after a protracted convalescent period but remain carriers. Atypical cases occur, some of which also give rise to the carrier state. Field's observations show that the carrier state can persist for at least 2 years. A point of public health importance is that small numbers of salmonella may be found in the milk of carrier animals.

A number of case records show that carrier cows transmit infection to calves *in utero* and *post natal*. Field states:

> It has been shown conclusively that transmission of infection from adult carrier animals to calves takes place. Although calves, recovered from infection do not, subsequently, excrete the organism in the faeces, it is probable that in some animals the organism localises in some organ or tissue. The examination of adult animals entering the abattoir for slaughter shows that *normal* cattle may be carrying Salm. dublin or Salm. typhi-murium. Salm. dublin has also been recovered from six out of thirty farm rats examined. Post-mortem examination of a carrier cow showed that the residual foci of infection included the gall bladder, spleen and all the lymph glands draining the liver and gastro-intestinal tract. Salm. dublin can survive for long periods in faeces and water. Faeces exposed on pasture has yielded the organism after seventy-three days in summer, while in contaminated water viable organisms were still present after eighty-seven days. [See also Clarenburg, Vink, and Schuumans (1950).]

Morrison, Ritchie, and Clayton (1951) made investigations into the salmonella carrier rate in domestic animals. Samples of faeces, bile, and liver swabs from slaughtered healthy cattle of Irish and non-Irish origin were examined for organisms of the salmonella group. *Salm. dublin* was isolated from 10 per cent of the faeces and 3·4 per cent of bile from the Irish cattle and 3·7 per cent and 0·5 per cent from the non-Irish cattle. Of 202 liver swabs from Irish cattle, 25 yielded *Salm. dublin*; of 171 non-Irish liver swabs none were positive.

Gordon (1951) reports the isolation of *Salm. dublin* from pigs which were suffering from dysentery. Murdock and Gordon (1953) examined 1,000 samples of faeces from apparently healthy cattle in Northern Ireland. An incidence of 8·6 per cent of *Salm. dublin* carriers as found.

HUMAN INFECTIONS WITH *Salm. Dublin*

Place	*Reference*	*Particulars*
Dublin	Bruce White, 1929	Pyelitis kidney and continued fever; single case.
Aberdeen	Smith & Scott, 1930	Three unconnected cases of continued fever. All positive blood cultures. All recovered.
Aberdeen	J. Smith, 1933	Three unconnected cases (2 infants, one 5 years); one septicaemia and mastoiditis, blood positive, fatal. One gastro-intestinal disturbance, blood negative, recovery. One meningitis, fatal; bacilli in cerebro-spinal fluid.
Aalborg (Denmark)	Grimsted, 1923	About 95 cases of acute gastro-enteritis at Aalborg Hospital. No deaths. Vehicle milk. Diseased cow which died and *B. paracoli* isolated from spleen and udder. Same organism in faeces of cases.
St. Pancras, London	Ministry of Health Report, 1928	Cases 22, no deaths. Vehicle junket. Suggested that was locally infected but information indefinite. *Dublin* isolated from faeces of cases.
Dundee, 1927	Tullock, 1939	About 280 cases of acute gastro-enteritis, no deaths. Vehicle milk. *Dublin* type from faeces and from internal organs of a diseased cow.
Wilton, 1936	Conybeare & Thornton, 1938	Over 100 cases of gastro-enteritis in children, no deaths. Vehicle milk. Faeces examined late and negative. Milk contained *dublin*, and this isolated from dung of cow with high titre.
S. Africa, 1938	Henning, 1938	Ten natives ate sick calf under-cooked. All suffered from food poisoning and one died. *Dublin* isolated from fatal case.
W. Riding of Yorkshire, 1943	Sutherland & Berger, 1944	162 cases (79 households), gastro-enteritis. No deaths. Vehicle milk. Cow apparently healthy, excreting *Salm. dublin* in faeces. At P.M. on animal, organisms isolated in pure culture from gall bladder and bowel.
Shropshire, 1947	Jones & Symons, 1948	32 cases gastro-enteritis notified. No deaths. Victims consumed cooked or raw sausages. *Salm. dublin* isolated from faeces and also from a sausage.
Aberdeen-shire (Kincardine O'Neil), 1947	Henderson, Michie, Rae & Smith, 1948	97 cases (32 families), acute gastro-enteritis. No deaths. Vehicle milk. Diseased cow, died from severe enteritis. *Salm. dublin* isolated from faeces of patients, the milk, material from dead cow, and from faeces of two calves.
Northern Ireland (Castle-derg)	Cromb & Murdock 1949	50 cases. Vehicle milk. Two animals excreting *Salm. dublin.*
Somerset (Chard)	McCall, 1953	610 cases. Vehicle T.T. raw milk. Gastro-enteritis. *Salm. dublin* isolated from milk, udder, and faeces of affected cow.

Edwards, Bruner, and Moran (1948) investigated the occurrence and distribution of salmonella types in the United States. During 1935–9, 12,331 salmonella cultures were examined. In all 105 types, 43 new types were discovered and 6 variants of certain types were identified. *Salm. typhi-murium* was the most frequently reported type and occurred in the widest range of animal species. This organism was on one occasion found in the faeces of a cow and from a number of persons who suffered from gastro-enteritis after drinking milk from the herd. *Salm. enteritidis* was frequent in rodents but rare in man. The dublin type was found only in the Western states. *Salm. cholerae-suis* was isolated not only from swine and man, but also from fowls, ruminants, carnivores, and rodents.

The following figures show the number of outbreaks and cases of salmonellosis recorded in the United States Public Health Reports (Public Health Service), during 5 years:

Year	*Outbreaks*	*Cases*
1952	31	1,335
1953	21	533
1954	26	1,164
1955	16	971
1956	16	1,952

Salmonellosis in Dogs, Cats, Birds, etc.

Shirlaw, McDonald, and Hayes (1946) record two authentic cases of salmonellosis in dogs which occurred in India. In one

The source was a cross-bred female terrier aged about four years and living in a hospital compound. The animal aborted after about a week's febrile illness and when first seen had an acute septic metritis with signs of septicaemia and threatening collapse. The metritis cleared up rapidly under treatment but the constitutional symptoms persisted for about three weeks and were accompanied by a short but severe attack of enteritis, with blood in the faeces, lasting about four days. Convalescence was protracted. Examination of the uterine pus revealed numerous coliform organisms, markedly phagocytosed. This organism was easily isolated by swabbing the uterine fundus and also from the faeces. The organism was found to be identical biochemically and serologically with *Salm. typhi-murium.*

Edwards, Hermann, Watt, De Capito, and Morn (1947) isolated 8 new salmonella strains from rectal swabs from cats, dogs, and chickens.

Wolff, Henderson and McCallum (1948) collected and examined rectal swabs from 100 dogs. The following salmonella types were isolated and identified from the stools of 18 of the dogs: *S.*

manhattan, *S. newport*, *S. minnesota* (both monophasic and diphasic varieties), *S. oranienburg*, *S. typhi-murium*, *S. bredeney*, *S. worthington*, *S. give*, *S. cubana*, *S. cerro*, *S. kentucky*, *S. illinois*, and *S. meleagridis*. The writers state:

> This work does indicate that the dog may be a frequent host for the Salmonella organisms. In view of the established pathogenicity for man of most of the above cited Salmonella organisms, the dog should be considered as a potential source of Salmonella infection in man.

FOOD POISONING OUTBREAKS IN ENGLAND AND WALES DURING THE FIVE YEARS 1953–57

Year	*Total number Outbreaks*	*Family Outbreaks*	*Sporadic Cases*	*Total All Incidents*	*Deaths*
1953	492	422	4,363	5,277	46
1954	506	630	4,880	6,016	29
1955	612	723	7,626	8,961	34
1956	563	616	6,534	7,713	44
1957	473	501	6,097	7,071	36

Compiled from Annual Reports on Food Poisoning contained in *Mon. Bull. Minist. Hlth. Lab. Serv.*

TYPES OF SALMONELLA ISOLATED FROM PRESUMED CAUSAL AGENTS OF FOOD POISONING IN ENGLAND AND WALES DURING THE FIVE YEARS 1953–57

Salmonella Types	1953	1954	1955	1956	1957	*Remarks*
Salm. typhi-murium	2,438	3,038	4,276	3,245	2,973	Types most commonly isolated
,, *enteritidis**	126	70	126	199	158	
,, *thompson*	84	83	164	83	79	
,, *newport*	50	35	66	97	88	
,, *dublin*	20	15	29	35	23	
,, *cholerae-suis*	13	11	18	11	19	
,, *bovis-morbificans*	81	26	32	25	58	
,, *derby*	11	10	42	50	20	
,, *stanley*	4	3	101	56	8	
,, *anatum*	30	20	50	78	75	
(*includes sub-strains)						
Salm. montevideo	18	13	9	3	9	
,, *oranienburg*	29	6	16	19	10	
,, *bareilly*	25	3	11	19	8	
,, *minnesota*	17	11	5	1	—	
,, *give*	8	2	6	8	13	
,, *tennessee*	15	8	4	6	6	
,, *bredeney*	3	20	14	55	52	
,, *heidelberg*	11	16	77	136	269	

Compiled from Annual Reports on Food Poisoning contained in *Mon. Bull. Minist. Hlth. Lab. Serv.*

Taylor (1949) reported the infection of a child with *Salm. typhi-murium* from a cat. The same worker identified *Salm. concord* from a case of gastro-enteritis in a child on a farm and from her cat. Bruner and Moran (1949) isolated 26 different types of salmonella organisms from dogs, of which the most frequent were *Salm. typhi-murium* (40 per cent of outbreaks), *Salm. cholerae-suis*, *Salm. oranienburg*, *Salm. newport*, *Salm. anatum*.

Cruickshank and W. Smith (1949) examined the faeces from 500 dogs, 500 cats, and 133 pigeons in London for salmonella. They found that 5 of the dogs (1 per cent), 7 of the cats (1·4 per cent), and 3 of the pigeons (2·25 per cent), were excreting salmonellae. The organisms from the dogs were *Salm. newport* (2), *Salm. typhi-murium* (2), and a salmonella of doubtful identity; from the cats, *Salm. typhi-murium* (3), *Salm. anatum* (2), *Salm. montevideo*, and *Salm. paratyphi* B; from the pigeons, *Salm. typhi-murium* (3). These organisms are all types known to be capable of causing disease in man. The investigators state that:

> The percentage of Salmonella excretors among dogs and cats is probably of the same order as that among rats and mice. The Salmonellae isolated are of species which have caused gastro-enteritis and in the case of Salm. para-typhi B, enteric fever in man. Further there are a number of instances in the literature in which infection of a person from a dog or cat has been established. It is therefore reasonable that the epidemiologist should consider the possibility that dog, cat and pigeon may be a reservoir of Salmonella infection from which human cases may occasionally arise.

See also Beekman and Bergsma (1950), Watt and De Capito (1950), Edwards and Fife (1951), Edwards, De Capito, and Fife (1951), Stucker, Galton, Edwards, and Fife (1951), Adler, Willers, and Levine (1951), Scatterday and Hardy (1952), Stucker, Galton, Cowderey, and Hardy (1952), McElrath, Galton, and Hardy (1952), Mackel, Galton, Gray, and Hardy (1952), Clarenberg, Teunissen, and Van der Ylerk (1954). Salmonella organisms have been isolated also from other animals and reptiles; see Rewell, Taylor, and Douglas (1948), Buttiaux and Gaumont (1948).

Steiniger (1954) discusses and gives details of his studies and investigations on salmonella infection of sea-birds and their eggs and points out that, parallel with these findings, an increasing number of salmonella infections in ornithologists and other bird-handlers have been reported.

Williams, Smith, and Buxton (1951) carried out a survey regarding the presence of salmonellae in the faeces of adult domestic animals in England and Wales. They found that:

Sixteen of 650 turkeys (2·5 %), two of 100 geese (2 %), six of ducks (1·2 %), four of 600 pigs (0·67 %), five of 700 chickens (0·67 %), three of 750 cows (0·4 %), one of 500 horses and none of 500 sheep were found to be excreting Salmonellae in their faeces. The organisms from the turkeys were Salm. typhi-murium (11), anatum (4), and tennessee; from the geese, Salm. typhi-murium and thompson; from the ducks Salm. typhi-murium (5) and meleagridis; from the pigs, Salm. meleagridis (3), and typhi-murium; from the chickens, Salm. typhi-murium (2), pullorum (2) and anatum; from the cows, Salm. dublin (3); from the horse, Salm. thompson.

No Salmonellae were isolated from the faeces of the following species of animals which were exhibited at national shows in this country; cows (430), chickens (420), ducks (155), and turkeys (63). All the Salmonella types that were isolated in the survey, with the possible exception of Salm. pullorum, are known to be capable of causing disease in man. [See also Sellers and Sinclair (1953); Schmid and Velaudopillai (1953).]

Pereira and Blaxland (1955) record outbreaks of *Salm. typhi-murium* infection among the birds on 2 large turkey farms. Seven human cases who had been in contact with the diseased birds also became infected.

Evolution of Salmonella Types

Savage (1956) remarks:

The enormous number of distinguishable Salmonella types, the wide range of animals invaded, and the varying types of infections caused form a complex picture. In my opinion the problems raised can best be linked, and in part explained, by considering them from an evolutionary standpoint.

He goes on :

Salm. typhi-murium is probably nearest to the common ancestor catholic in its distribution and undifferentiated in its host selections. Specialism has enabled some strains to attain a high measure of host specificity. *Salm. paratyphi* B has become for all practical purposes a human invasive parasite causing long-continued infection and rarely gastro-intestinal symptoms. It is no longer invasive to the lower animal by natural infection. The general picture of paratyphoid fever is, however, very definite, and not a single outbreak of food poisoning in the long records of the Public Health Laboratory Service has been ascribed to *Salm. paratyphi* B. Other strains with a high degree of host specialisation are *Salm. abortus-ovis* for sheep, *Salm. gallinarum* and *Salm. pullorum* for fowls, and *Salm. abortus-equi* for horses. *Salm. dublin* is particularly interesting, for it tends to limit itself to calves and cattle but so far very incompletely, and this characteristic is accompanied by varied patterns of pathological changes which emphasise its stability.

The differentiation of salmonella strains is almost entirely based upon antigenic differences and under ordinary conditions

these remain stable. This of course does not exclude the conception of their evolutionary origin, both as regards their antigenic structure and their animal distribution. There is indeed considerable evidence in favour of such a hypothesis and is in line with general biological tendencies. Recently this hypothesis is being strongly re-enforced by the fact that salmonella type changes have been achieved in the laboratory, for example *Salm. oranienburg* has been changed into *Salm. montevideo*, although as yet the change has not been reversed. Three laboratory procedures are being used, i.e. growing the organism in anti-serum, the method of gene recombination (essentially a sexual process in bacteria) and action on the organism by a selected bacteriophage. (Savage, private communication.)

Toxins

In recent years evidence has accumulated, showing that many outbreaks of food poisoning have been due to undestroyed poisonous substances elaborated by certain organisms. This may have been due to the fact that bacteriological research failed to reveal the presence of the causative organisms. The extremely short incubation period (2 to 4 hours or even less) together with the very acute symptoms, suggested the action of a preformed toxin in the food ingested, especially in the case of canned foods.

Topley and Wilson (1936) remark:

> It was supposed that the organisms had multiplied in the food prior to its consumption and had formed thermostable toxic substances. The subsequent cooking to which the food was exposed destroyed the organisms themselves, but did not seriously affect their toxic products, which were therefore able to give rise to food poisoning on ingestion. No adequate confirmatory evidence of the formation of specific exotoxins by members of the Salmonella group was forthcoming and the balance of evidence appeared to be against this view. . . . Summarising, we may say that evidence has been accumulating in the past few years to show that many of the 'toxin' outbreaks of food poisoning are due to the production of toxic substances in the food prior to its consumption. These substances, the exact nature of which is still unknown, are formed under suitable conditions by a number of different bacteria, of which staphylococci, streptococci, coliform, Proteus, and possibly Salmonella, organisms appear to be the most important.

Savage (1920, 1923, and 1932) advanced the toxin theory, and Savage and Bruce White (1925), referring to outbreaks due to the presence of undestroyed salmonella group toxins, stated

> These form a very important group, particularly in relation to canned foods. It will readily be appreciated that the furnishing of

complete, or even presumptive, proof that these toxins are the cause of any outbreak is a matter of great difficulty. There are no living bacilli to isolate. Our studies on this point have been to a certain extent progressive, and for the later outbreaks improved methods have enabled us to furnish proofs of a more conclusive nature than we were able to do for many of the earlier outbreaks.

The toxin hypothesis has at times created much controversy in this country and abroad, doubtless due in a measure to the fact that there is no simple definite laboratory test to demonstrate the presence of a preformed toxin in suspected foodstuffs, or in broth cultures prepared from them. Little is known of the exact nature and mode of origin of these poisonous thermostable water-soluble substances as to whether they are specific bodies elaborated by bacteria or whether they represent products of dead organisms. As shown by many observers these substances are not always poisonous to human beings or to experimental animals.

Savage and Bruce White (1925) studied the methods of action of strains of the salmonella group upon the alimentary tract and demonstrated the presence of a powerful irritant, both in boiled and unboiled cultures, which acts rapidly and intensively upon the mucous membrane of the stomach of young rabbits and was most readily demonstrated in those types within the group which were responsible for food poisoning. Later experiments showed that it was possible to produce toxic effects upon mice when fed with salmonella strains grown in certain media.

Other investigators, Geiger and Meyer (1928) and Branham, Robey, and Day (1928), also found that boiled salmonella cultures when fed to mice proved fatal. Dack, Harmon, and Jarra (1928), however, obtained negative results (with one exception) on introducing heat-killed cultures of salmonella into the internal tract of monkeys and other animals.

Dack, Cary, and Harmon (1928), failed in an attempt to produce intoxication in human volunteers by feeding them with heat-killed cultures and filtrates of salmonella organisms (*Salm. aertrycke* and *Salm. enteritidis*). Similar cultures and filtrates when injected intravenously into rabbits produced the characteristic symptoms and death. It would appear, however, that in these experiments freshly isolated strains of salmonella organisms were not used. Dack, Jordan, and Wood (1929) fed living salmonella cultures (*Salm. aertrycke* and *Salm. enteritidis*) to rhesus monkeys causing diarrhoea, malaise, and loss of appetite. The specific bacilli were not found in the blood stream. Monkeys fed with

equivalent heat-killed portions of the same suspension exhibited no symptoms. In 1930, Elkeles reviewed the whole subject of these animal experiments.

It may be of interest to mention the following outbreak, which occurred at Edinburgh in 1926, as illustrating this type of toxin poisoning. Three persons consumed a mutton stew and were attacked, after a very short incubation period, with acute food-poisoning symptoms. From the stew no 'living Salmonella bacilli' could be isolated, but from part of the mutton not used to make the stew, a salmonella strain was isolated which was pathogenic to guinea-pigs.

Many similar toxin type outbreaks have been recorded from time to time in this country. Dolman and other observers showed that certain strains of staphylococci produce an enterotoxin which is a cause of food poisoning. Whether certain strains of other pathogenic organisms (including those of the salmonella group) under favourable conditions elaborate these thermostable entero-toxic substances and so cause the toxic type of food poisoning, can only be proved by further experimental researches.

Boivin and Mesrobeanu (1933–5), Raistrick and Topley (1934), and Morgan (1937) described a method for extracting these thermostable substances and showed they consisted of lipopoly-saccharide. When injected intravenously into rabbits they give rise to weakness and prostration sometimes accompanied by tremors and diarrhoea, according to Delafield (1934). Injected intraperitoneally into mice in a dose of about 0·5 mg., they caused death, according to Martin (1934).

Dolman (1943), in discussing the literature and experimental work bearing on the conception of a toxin type of salmonella food poisoning, remarks:

> To summarise, although a far greater number of negative human feeding experiments, involving a wider variety of types of Salmonella, and using freshly isolated strains, would need to be carried out before the categorical claim could be made that a 'toxin' type of Salmonella food poisoning cannot occur, the available evidence to date does not suggest that Savage's hypothesis accounts for any significant proportion of such outbreaks. Savage himself foresaw that an alternative conception might eventually be found to fit the facts more satisfactorily.

Dack (1944) states:

> The evidence at the present time is overwhelmingly in favour of the view that Salmonella food poisoning is actually an infection and that toxins or toxic substances play no rôle.

French physicians continue to maintain that toxic infectious factors are both involved in the acute, violent, gastro-intestinal attacks, especially when meat is the incriminated vehicle.

Resistance to Heat

The remarkable heat-stable properties of these poisonous substances (Cathcart found that *B. enteritidis* toxin withstood heating to 100°C. for 30 minutes) have considerable bearing on the processing of canned foods, especially in the United States where the subject has been under active investigation, on account of its importance to the food preservation industry.

Savage (1932) called attention to:

> The close association of this type of food poisoning with canned foods—that is, foods strongly heated after they are put into the tin. The temperatures used (100°C. or above) are adequate to kill non-sporing bacilli, but Salmonella toxins can survive these temperatures. Assuming specific infection, before canning, of a portion of the food, the conditions actually found—that is, a food perfectly sound physically, freedom from living pathogenic bacilli, the presence of resistant toxins, —are just those one would expect.

A considerable amount of research and experimental work has been carried out in this country, in America and in Germany to ascertain the thermal destruction point of toxins. This varies through a wide range of temperatures and is dependent, moreover on several factors, including the character; (size of food particles) and composition of the contents of the can, the hydrogen-ion concentration and the nature of the toxin itself. In canned foods the heat penetrates to the centre of the contents by convection and conduction, but the character of the food greatly affects the convection currents. In thick and solid substances such as meat, the heat, being by conduction, penetrates very slowly. Therefore it is necessary that canned foods should be submitted to a sufficiently high temperature for the required length of time to be certain of the destruction of any bacterial toxins that might be present. It is also essential in processing that no over-heating takes place, otherwise the food may be spoiled.

In this connection some interesting investigations have demonstrated that meat, in particular, is a poor conductor of heat.

Perroncito studied the heat penetration of a boiled ham. A ham of about 6 kilos weight (13 lb.) was placed in cold water which was raised to boiling-point. The water boiled when the interior of the ham was only 25°C. (77°F.). After 35 minutes the temperature

was 35°–40°C. (95°–104°F.). After 2 hours the temperature in different parts of the interior was 46°, 55°, 58°, 62°, 64°, and 67°C. (152·6°F.). A larger ham, weighing about 8 kilos (16 lb.), treated in the same way, only showed an interior temperature of 44·5°C. after $2\frac{1}{2}$ hours, while after $3\frac{1}{2}$ hours the temperature varied from 62°–84°C. (183·2°F.) in different parts.

Beveridge and Fawcus (1908) carried out some experiments on the penetration of heat into the substance of meat in cans. They found that when a tin was simply boiled in water, the centre of the meat did not reach 100°C. even after 5 hours, and that with higher temperatures, the undermentioned results were recorded:

Outside Temperature	*Size of Tin*	*Time Taken by Central Thermometer to Reach*		*Number of Experiments*
		100°C.	105°C.	
		minutes	minutes	Average of
107°C.	1 lb.	58	80	5
107°C.	2 lb.	95	123	5
120°C.	1 lb.	22	24·4	5
120°C.	2 lb.	28	36·2	5
130°C.	2 lb.	17	22	2

With larger tins the rate and time of heat penetration would be considerably longer.

In liquid foods, such as soups and beverages, the heat being by convection, penetrates rapidly and, providing they are boiled for a sufficient length of time, destroys infectivity. This is well illustrated in an outbreak at Newcastle (1913) where 523 people consumed milk infected by *B. enteritidis*, and not a single person who drank the milk after it had been boiled was infected.

Brightwell (1954) found that, in meat pies weighing 6 lb. 8 oz., the temperature in the centre did not reach boiling point until the pie had been heated to 177°–204°C. for $2\frac{1}{2}$ to 3 hours.

Miller, Nicol, and Ramsden (1955) observed that salmonellae were killed in the centre of meat pies weighing 4 oz. after they had been heated to 160°C. for 60 minutes or 193°C. for 40 minutes.

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SALMONELLA SEROTYPES ISOLATED BY OBSERVERS IN ENGLAND AND WALES 1920–57

Type and Antigenic Structure	*Reference*
Salm. aberdeen XI: i–1, 2, 3.	Smith, J. (1934): *J. Hyg., Camb.*, **34**, 351.

Type and Antigenic Structure	*Reference*
Salm. birkenhead VI, VII: c–1, 6.	Taylor and Douglas (1948): *J. Clin. Path.*, **1**, 237.
Salm. bolton III, X: y–e, n, Z_{15}.	Greenwood, Powis, Douglas and Taylor (1953): *Mon. Bull. Minist. Hlth. Lab. Serv.*, **12**, 26.
Salm. brancester I, IV, XII: Z_{29}.	Macdonald et al. (1948): *Mon. Bull. Minist. Hlth. Lab. Serv.*, **7**, 158.
Salm. bury (4, 27, 12: c–Z_6)	Stitt and Walsh (1956): *Int. Bull. Bact. Nomen. & Tax.*, **6** (No. 2), 51.
Salm. cambridge III, XV: e, h, –1, w.	Wilson, Taylor, and Anderson (1947): *Mon. Bull. Minist. Hlth. Lab. Serv.*, **6**, 135.
Salm. cardiff VI, VII: k–1, 10.	Taylor, Edwards, and Edwards (1945): *Brit. Med. J.*, **1**, 368.
Salm. clerkenwell III, X: 1, w–z.	Story et al. (1952): *Mon. Bull. Minist. Hlth. Lab. Serv.*, **11**, 23.
Salm. colindale 6, 7, : 5 ; 1, 7.	Price and Holt (1955): *Int. Bull. Bact. Nomen. & Tax.*, **5**, 1.
Salm. derby (I), IV, XII: f, g –.	Peckham (1923): *J. Hyg., Camb.*, **22**, 69.
Salm. derby (I), IV, XII: f, g –.	Savage and Bruce White (1925): *Spec. Rep. Ser. Med. Res. Counc., Lond.*, No. 91.
Salm. dublin I, IX, XII: g, p –.	Bruce White (1929–30): *J. Hyg., Camb.*, **29**, 443.
Salm. eastbourne (I), IX, XII: e, h–1, 5.	Leslie and Shera (1931): *J. Path. Bact.*, **34**, 533.
Salm. gloucester 1, 4, 12 (27); i: 1, w.	Waite and Taylor (1957): *Mon. Bull. Minist. Hlth. Lab. Serv.*, **16**, 14.
Salm. hull (XVI: b: 1, 2 –)	Alexander, Douglas, and Taylor (1954): *Mon. Bull. Minist. Hlth. Lab. Serv.*, **13**, 117.
Salm. london III, X: i, v–1, 6.	Bruce White (1926): *Spec. Rep. Ser. Med. Res. Counc., Lond.*, No. 103.
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Salm. menston VI, VII: g, s, t –.	Colbeck, Douglas, and Taylor (1951): *J. Path. Bact.*, **63**, 754.
Salm. neasden IX, XII: g, s, (t)–e, n, x.	Douglas, Taylor, and McMath (1951): *Mon. Bull. Minist. Hlth. Lab. Serv.*, **10**, 250.
Salm. newlands III, X: e, h–e, n, x.	Greening, Price, and Taylor (1953): *Mon. Bull. Minist. Hlth. Lab. Serv.*, **12**, 89.
Salm. newport VI, VIII: e, h, – 1, 2, 3.	Schutze (1920): *Lancet*, **1**, 93. Bruce White (1926): *Spec. Rep. Ser. Med. Res. Counc., Lond.*, No. 103.

Type and Antigenic Structure	*Reference*
Salm. norwich VI, VII: e, h, – 1, 6.	Taylor, Macdonald, and Sivell (1951): *Mon. Bull. Minist. Hlth. Lab. Serv.*, **10**, 76.
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Salm. poona XIII, XXII: z–1, 6.	Bridges and Scott (1935): *J. R. Army Med. Cps.*, **65**, 221.
Salm. reading IV, XII: e, h–1, 5.	Schutze (1920): *Lancet*, **1**, 93.
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Salm. stanley IV, V, XII: d–1, 2.	Schutze (1920): *Lancet*, **1**, 93. Savage and Bruce White (1925): *Spec. Rep. Ser. Med. Res. Counc., Lond.*, No. 91.
Salm. thompson VI, VII: k–1, 5 or 1, 10.	Scott (1926): *J. Hyg., Camb.*, **25**, 398.

Chapter IV

SEASONAL PREVALENCE, INCUBATION PERIOD, CLINICAL FEATURES AND SYMPTOMS, POST-MORTEM SIGNS, MORTALITY

Seasonal Prevalence

The seasonal variation of food poisoning due to salmonella organisms is probably a result of the rise in temperatures during the summer months, which may favour their rapid multiplication in infected foods, thus producing the moderately large dose

SEASONAL INCIDENCE OF OUTBREAKS AND RELATED OUTBREAKS DURING 1955

Month	*Salmonellae*	*Staphylococci*	*Cl. Welchii*	*Other organisms*	*Chemical*	*Not discovered*	*All outbreaks*
January	46	2	2	1	—	6	57
February	23	1	4	—	—	6	34
March	28	1	3	1	—	3	36
April	43	—	4	—	—	6	53
May	44	—	4	—	—	4	52
June	51	10	2	—	—	9	72
July	99	13	14	—	—	7	133
August	95	18	6	1	—	10	130
September	103	9	10	—	1	9	132
October	47	3	16	—	—	10	76
November	35	3	11	—	—	15	64
December	13	3	7	—	1	6	30
	627	63	83	3	2	91	869

Compiled from Annual Reports on Food Poisoning contained in *Mon. Bull. Minist. Hlth. Lab. Serv.*

necessary to cause the typical gastro-intestinal symptoms. It is possible that salmonella organisms have greater virulence in warm weather and that susceptibility to infection by some human beings is by increased sensitiveness of the alimentary tract during these warmer periods. It has been suggested that the seasonal incidence depends to some extent on the proportions of canned, preserved, and reheated foods to fresh foods consumed. According to recent

statistics, the highest incidence occurs between the months of June and October with 'peak' in August-September.

Incubation Period

No manifestations occur during the incubation period (i.e. the interval between the consumption of the incriminated food and the onset of the symptoms) which varies considerably and may range from 3 to 36 hours, but averages from 3 to 24 hours. These time variations may be influenced by the following:

(1) Infection Type of Food Poisoning. The ingested food may contain living bacilli only, and in consequence a definite incubation period (3 to 24 hours) must elapse during which the organisms develop sufficient poisonous substances to produce the typical symptoms. The incubation period is influenced also by the amount of food consumed and the degree of bacterial contamination.

Savage (1957) is of opinion that:

> when a food infected with a living salmonella is eaten the food usually contains a mixture of living bacteria and some already produced toxin. The amount of the pre-formed toxin will depend upon the nutrient quality of the food for bacterial growth, the time interval from its initial infection and the temperature at which the food is kept. The amount of pre-formed toxin affects the length of the period between the meal and the onset of symptoms. Consequently this may vary from some 7 hours up to 30 hours or so. About 18 to 24 hours is a common period. If the ingested bacilli continue to produce toxin this may extend the duration of symptoms over several days.

(2) Toxin Type of Food Poisoning. The ingested food may contain preformed heat-resisting toxins. In which case, the incubation period will be short (1 to 6 hours, usually 1 to 3 hours) as the poisonous substances produce the symptoms rapidly and more markedly.

Clinical Features and Symptoms

There is distinct uniformity in the clinical features in all cases of bacterial food poisoning. The symptoms, however, vary in duration and intensity in different outbreaks.

Illness of the *infection* type is sudden in onset and usually commences with nausea (with or without headache) and abdominal pain. The characteristic symptoms in typical cases are nausea and gastro-intestinal disturbance; this may be only simple diarrhoea (with or without headache or sickness) or severe inflammation of the alimentary tract (persistent diarrhoea) accompanied by

retching and vomiting. There may be intense abdominal pain and sometimes cramps. Marked prostration is a characteristic feature of the illness and may persist long into convalescence. In chronic cases the diarrhoea is severe with repeated offensive motions. The stools are watery, greenish in colour, and sometimes tinged with blood. There may be fever (temperature up to 100°–102°F.). The tongue becomes coated and breath is offensive. In some instances cold sweats, rigors, giddiness, and pains in the back and limbs occur. Herpes or urticarial rashes are not uncommon and occasionally eye symptoms (pupil irregularity) have been recorded.

In severe illness there is restlessness with great thirst (dehydration). The organisms may invade the blood stream and give rise to infection of the heart or membranes of the brain and spinal cord, which, in extreme cases, may be followed by coma and death. The latter occurs more frequently in young or elderly patients. In ordinary cases, however, the symptoms gradually diminish after 24 to 48 hours, but general weakness may persist for days or even weeks. Some cases bear a close resemblance to paratyphoid fever, except that recovery is rapid.

Post-Mortem Signs

The changes seen at the autopsy are slight compared with the severity of the illness. As a rule, the mucous membrane of the stomach and intestines is swollen and congested, the latter showing minute haemorrhages. Cloudy swellings are noticeable in the kidneys and the spleen is enlarged. Pale yellowish areas or spots are observed on the liver, which microscopic examination shows is due to intense fatty degeneration of the cells. The causative organisms can be isolated from the heart's blood, spleen, and other viscera.

Mortality

The mortality rate due to salmonella food poisoning is distinctly low, but varies in different outbreaks in this country and abroad. In the 112 outbreaks with 9,190 cases which occurred in England and were recorded by Savage (1920), it was 1·5 per cent. Savage and Bruce White (1925) found the mortality rate in milk outbreaks alone to be less than 0·2 per cent.

In the United States, Geiger (1923) estimated it at less than 0·5 per cent, but Feig (1950) gives 3·6 per cent. In Germany, Meyer

(1913) recorded a death rate of 1·0 per cent in outbreaks due to salmonellosis. Lentz (1924) gives 4·5 per cent.

In England and Wales in recent years it would appear that in outbreaks of food poisoning due to salmonella infections the mortality rate averages from 0·4 to 1·3 per cent.

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Chapter V

FOODS AS VEHICLES OF INFECTION

The suitability of a food for the multiplication of food poisoning organisms is a factor of considerable significance. In England and Wales, between the years 1941 and 1955, 2,346 outbreaks and family outbreaks of food poisoning were reported in which it was possible to establish definitely the vehicles of infection.

Meat (fresh, canned, processed, and made-up, soups and gravies) was implicated in 1,688 outbreaks (72 per cent), of which 1,315 (78 per cent) were associated with processed and made-up meats. Meat and meat products appear to figure less prominently in other countries. In the United States of America during 1945-7, of the 392 cases reported to the Public Health Service and caused by a single organism, 127 (32 per cent) were attributed to meat or meat products.

While infection of food may sometimes prove to be of animal origin (e.g. salmonellosis), more often the human bowel (salmonellae, shigellae), skin, nose, throat, infected injuries (staphylococci) are frequent sources in England and Wales. *Cl. welchii* infections may arise either from a human or animal source. This group of organisms is heat-resistant and chiefly associated with re-heated meat dishes. Soil organisms (*Cl. botulinum*) are very rarely implicated in food-poisoning outbreaks in Great Britain.

It is not possible to be dogmatic about 'sources of infection' since excretors of causative organisms in food poisoning cases may be from temporary or symptomless carriers, though strains or phage types may have particular associations which help to differentiate the probable origins. Here, however, we are less concerned with the probable origin of infective organisms than with the actual role of various foods acting as vehicles of infection. Nevertheless the susceptibility of a food to act as a means of infection will be related both to the possible source from which organisms gain access and to the degree to which handling and processing contribute to the proliferation of bacteria or the development of toxins.

Meat and Meat Products

The risk of meat becoming infected intravitally, for instance, means not only that fresh meat and offal may themselves

occasionally act as vehicles—often depending partly on slaughter-house hygiene, partly on storage and cooking temperatures—but also that the introduction of a few organisms from lightly infected meat from which products are to be manufactured can easily act as 'starters' for large-scale bacterial activity. The earlier recorded cases of meat poisoning appear to have been of this type, and often the history has shown association with 'emergency slaughter'.

Meyer (1943) recorded 32 outbreaks of meat poisoning in Germany for the previous year, in which meat from emergency-slaughtered animals caused 74 per cent of the 1,357 illnesses and 40 per cent of the 15 deaths.

More commonly, however, meat and meat products serve as vehicles of exogenous contamination, i.e. acquiring infection from human and environmental sources. In this role the extent of manipulation, physical conditions of processing, and specific practices are of the utmost importance.

The Manufactured Meat Products Working Party's Report (1950) pointed out the significance of hand-skinning ox tongues without further submission to heat-treatment. Gelatine was similarly discussed, especially in relation to the sub-lethal temperatures at which handled. Mincing and grinding, in the manufacture of sausages and similar products, not only ensures a wide dissemination of any chance contaminent but, by raising the temperature of the mix, may provide favourable incubation conditions.

Meat Pies

The manufacture of meat pies may combine a number of these hazards—manipulation, distribution of infective material, incubation conditions started off by mincing and maintained even heat through the ovens, and finally the introduction of ideal bacterial pabulum in the jellying process. Indeed an unscientific reliance on 'heat-treatment' in a particular process may lead to unwarranted liberties in the preliminary processes. When the 'heat-treatment' itself is unreliable, bacteriologically, disaster may result.

Delepine and Howarth (1902) showed that the internal temperatures of apparently well-baked meat pies might not exceed 47·2°C., and that an over-baked pie only attained an internal temperature of 86·6°C. How serious are these hazards even in a well-conducted bakery was shown by a food-poisoning outbreak which occurred in south Lancashire in 1953 (Miller, Nichol, and Ramsden, 1955), where 1,150 persons were infected by *Salm. bovis*

morbificans conveyed by meat pies. Bacteriologically intravital infection of the meat was not demonstrated but there is a strong presumption. Of more importance was the observation that *Salm. bovis morbificans*, when experimentally inoculated into 6 oz. and 12 oz. pies, survived a temperature of 320°F. after 50 minutes baking in sufficient numbers to ensure gross infection when gelatine was added. Exposure to 380°F. for 60 minutes, however, proved lethal. Bakery oven records showed that the cooking times and temperatures were insufficient to kill the organisms present in the pies. Hand pouring of warm gelatine would always be a hazard in the event of human carriers being so employed. Indeed it was thought that two employees engaged in pie jellying (but normally occupied in gut washing and weighing raw meat) were carriers of *Salm. minnesota* which was responsible for the illness of some 500 persons who consumed pork pies made in a Midland county (McCracken, 1954). This organism was recovered from one sample of imported frozen pork collected from the factory and also from swabs taken from the cutting bench where the pork was unpacked.

Mince

This meat-food is frequently implicated in food poisoning outbreaks partly for reasons already indicated and partly because there is a tendency to regard it as an easily cooked type of meat and either it is presented once-cooked at insufficient temperatures, or after partial cooking on one day it is reheated for consumption the next day, in the interval slow cooking having served to incubate any organisms which have survived the initial par-boiling. Where human carriers are concerned, the time and temperature of final cooking are the ultimate defence.

An outbreak of gastro-intestinal illness at a girls' school in Yorkshire in 1951 illustrates this point (Dolman, 1957). Shepherd's pie was served to 439 girls; 63 clinical cases of paratyphoid fever developed and 47 apparently healthy children were found to be excreting *Salm. paratyphosus* B. Fresh mince had been stewed in water, separated from the stock and both refrigerated overnight. The next morning dishes containing 50 helpings of mince, stock, onion, and potatoes were cooked for times varying between 25 and 60 minutes and served to the girls at three sittings. Nearly all the cases ate the food at the first sitting but there were no cases from the last sitting. The probable source of infection was the cook who

was found positive serologically; she ate the food after the third sitting.

Salmonellosis of Animal Origin

Dolman (1957) draws a distinction between infections arising from meat or meat products from human or environmental sources, in which he characterizes as 'food poisoning', and those arising from an infection in life of the animal which he classifies as 'zoonoses acquired chiefly through the intestinal tract'. Such a distinction has not yet been made in this country and the measure of risk, so far, has not been fully assessed.

Smith and Buxton (1951) found salmonellae in the faeces of domestic animals on the following scale:

	per cent.
Turkey	2·50
Duck	1·20
Chicken	0·67
Horse	0·20
Goose	2·0
Pig	0·67
Cow	0·40

Intravital infection of food animals may be on a lighter scale than for instance in the United States, where Felsenfield, Young, and Yoshimura (1950) found salmonellae in foods purchased in the open market in Illinois in the following percentages:

	per cent.
Poultry (U.S. inspected)	1·20
Poultry (uninspected)	9·20
Hen's eggs	0·60
Egg powder	3·00
Pork (U.S. inspected)	12·60
Pork (uninspected)	27·00
Beef (U.S. inspected)	0·20
Hamburger meat	18·00

Recently in Florida 23 per cent of samples of pork sausage and 12·5 per cent samples of smoked pork sausage revealed salmonellae (Galton, Lowery, and Hardy, 1954).

The Essex outbreak (Camps, 1947) of *Salm. typhi-murium* originating in one or two infected pigs sent for slaughter and spread by unhygienic practices is by now a classic example of the potential hazards from zoonobic salmonellosis.

Lundbeck, Plazikowski, and Silverstolpe (1955) record an even more unfortunate example of these hazards when things go wrong: overloaded slaughter-house, refrigeration failure, high atmospheric temperature, which together with intravital infection by *Salm.*

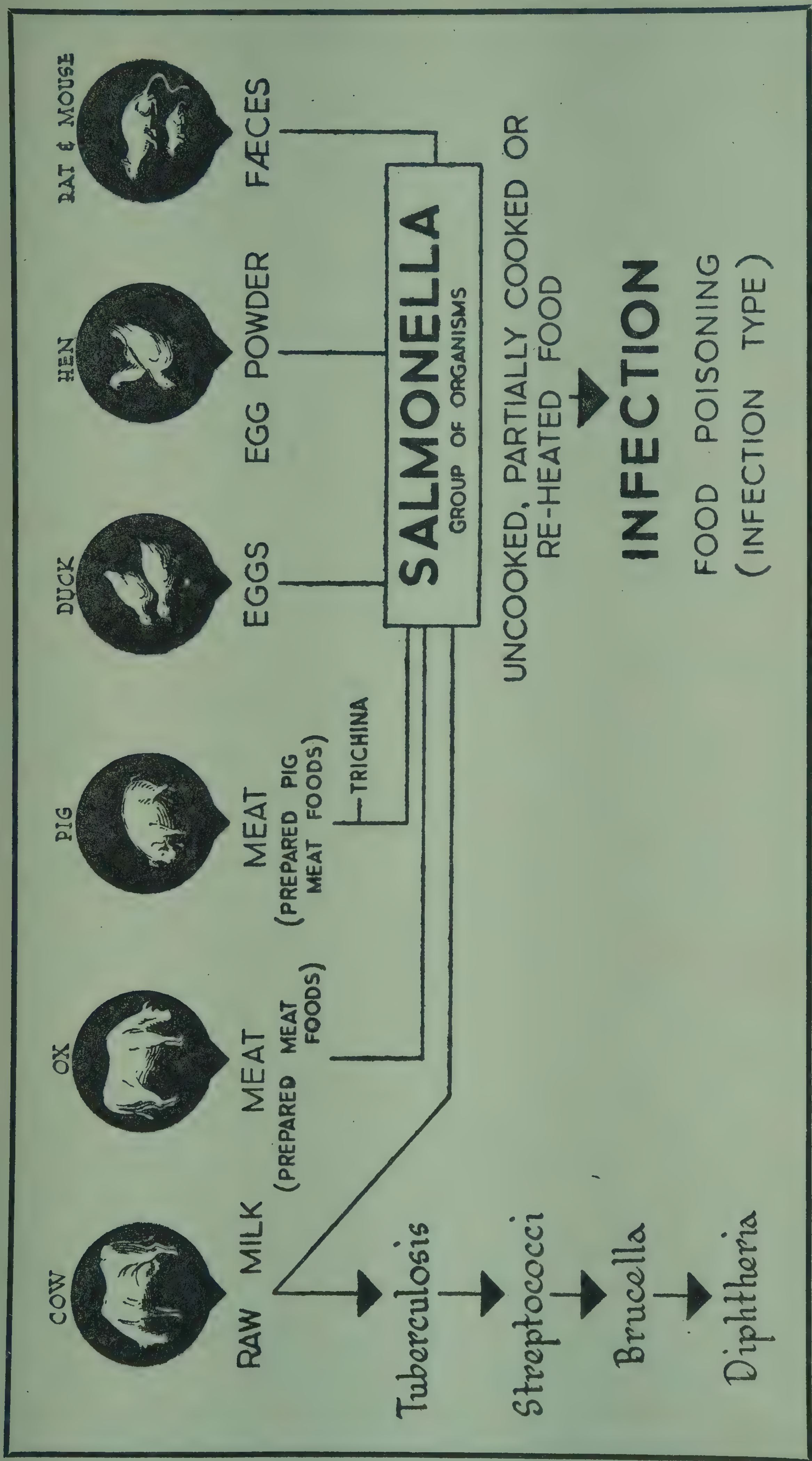

Fig. 1. Animal Reservoirs of Food-borne Infection.

typhi-murium gave rise to the largest recorded outbreak of this kind in Sweden where 7,717 cases were notified with 90 deaths.

Meat Products, Toxin Type

The following table shows that meat or meat products play an even greater part as vehicles in the toxin type of food poisoning, e.g. staphylococcal.

FOOD POISONING IN ENGLAND AND WALES 1953–55

Outbreaks (including family outbreaks) associated with processed or made-up meats

Type of Meat Product	*Presumed causal organisms*					*Total*
	Salmonellae	*Staphylococci*	*Cl. welchii*	*Other organisms*	*Not discovered*	
Meat pies and pastries	12	13	34	4	41	104
Reheated meat	2	9	52	2	35	100
Cold meat	17	14	21	2	20	74
Ham and boiled bacon	2	43	—	1	15	61
Pressed meats	10	27	3	1	14	55
Brawn, meat in gelatine, meat roll	7	17	7	4	17	52
Sausages, liver sausages, etc.	10	5	1	—	14	30
Stews, made-up dishes (including mince)	5	6	9	—	9	29
Sandwiches	3	10	—	—	7	20
Tongue	1	7	1	—	6	15
Corned beef, luncheon meat	—	2	—	—	4	6
Stuffed meat	2	1	1	—	1	5
Bath chaps	—	5	—	—	—	5
Various	2	2	—	—	2	6
Total, all types	73	161	129	14	185	562

(Compiled from Annual Reports on Food Poisoning contained in *Mon. Bull. Minist. Hlth. Lab. Serv.*)

Darling (1942) records the following interesting and classic case of staphylococcal intoxication via meat gravies, soups, etc. A woman (61 years old) while suffering from a whitlow of the thumb opened a tin of soup and partook of some of the contents. There were no ill effects and the thumb healed. Six days later she decided to finish the soup which had been kept in the warm kitchen;

within 3 hours after consuming the warmed-up soup she was taken ill with vomiting and diarrhoea; admitted moribund to hospital 22 hours later and died shortly afterwards. Coagulase positive haemolytic *Staphylococcus aureus* were isolated from the alimentary contents and also from the inside of the soup tin.

Milk

Staphylococcus aureus has been implicated in most milk-borne food poisoning outbreaks recorded during 1951–5 and is usually associated with mastitis in the cow.

Eggs

Salm. typhi-murium features with great regularity as the causative organism in egg-borne food poisoning, as shown in the following table:

1949	38 per cent of all *Salm. typhi-murium* outbreaks.
1950	32 per cent.
1951–2	38 per cent (33 of 87; 28 involving duck eggs).
1953	27 per cent (21 of 77; 18 involving duck eggs).
1954	32 per cent (10 of 31; all duck eggs).
1955	7 per cent (2 of 27; duck eggs).
Also	1 *Salm. pullorum*
	1 *Salm. thompson* and *Salm. paratyphi* B—Chinese egg.

Egg Products

In the *Monthly Bulletin of the Ministry of Health and the Public Health Laboratory Service*, 1956, 'Food Poisoning in England and Wales, 1955', it states:

It is well known among those interested in public health that Chinese dried egg albumen, Chinese frozen whole egg and Chinese frozen egg albumen have frequently been found to be contaminated not only with salmonellae of the food-poisoning types but also sometimes with *Salm. paratyphi* B. Semple, Davies and Parry (1956) reported . . . salmonellae from 14 per cent of 1,625 samples of dried Chinese albumen and that in one shipment 47 per cent of samples were contaminated. It was reported in the British Medical Journal (Annotation 1955) that 15 to 75 per cent of samples of Chinese dried albumen were contaminated with salmonellae and that 11 strains of *Salm. paratyphi* B. had been isolated from it. In addition salmonellae including *Salm. paratyphi* B. were isolated from Chinese frozen whole egg and frozen egg albumen. Egg products from home sources and from countries other than China have also been found to be contaminated with salmonellae but not so far as is known with *Salm. paratyphi* B. . . . Foods such as imitation cream can readily be contaminated from infected egg products in a bakery—for example via utensils or washing-up water. Two-thirds of

the strains of salmonellae isolated from Chinese egg products in a recent survey made by the Public Health Laboratory Service were identified as *Salm. thompson*. In human infections in England and Wales *Salm. thompson* is the second most common type. Though first isolated from cases as long ago as 1924 (Scott 1926) the source has always been a mystery. It is not a common type in cattle, sheep, pigs, rats or mice, and is only occasionally met with in fowls in Great Britain. The number of strains isolated fell during the war years when no Chinese egg products were imported, and rose again when imports were resumed. These products therefore—which have been coming into the country since 1921—may well be the main reservoir of this particular *Salmonella*.

Knowles (1953) recorded the following microbiological observations on liquid raw egg:

Liquid raw hen egg	244 samples	4·2 per cent positive 6 *Salm. typhi-murium* 3 *Salm. thompson*
Liquid raw duck egg	49 samples	67·40 per cent positive 33 *Salm. typhi-murium*

Fish, Fruit, and Vegetables

Foods of this kind appear in a relatively minor role as vehicles of food-borne infections. Collectively canned items appear rather more frequently in association with staphylococcal types, but the relative infrequency of occurrences renders any conclusions doubtful. It has been suggested, for instance, that canned foods may become infected by staphylococci during cooling, as a result of strained seams, but contamination from human sources of contents after opening the can is an equally tenable hypothesis.

Shell-Fish

In recent years the role of shell-fish as a vehicle of food poisoning has appeared to be a declining one. In 14 outbreaks 1951–5, the organism responsible was identified on three occasions only—a much higher 'undiscovered' rate than for any other food. In general, failure to discover an organism is probably due in a measure to absence or delay in investigations or to belated notifications. Whether the past 'bad name' of shell-fish has attracted more odium than it deserves is a matter of speculation.

Physical Appearance of Incriminated Food

With the exception of *Cl. botulinum*, the pathogenic organisms responsible rarely, if ever, effect any recognizable change in the physical appearance, taste or odour of the foods which act as

vehicles of infection. Recognizable changes in appearance of the food are usually associated with organisms which cause decomposition and do not appear to be incriminated in food poisoning outbreaks. The changed appearance and odour usually effectively prevents consumption in such cases.

The inability to recognize physically foods which are contaminated by pathogenic organisms was tragically illustrated by an incident related by Van Ermengem (1896), which occurred in Moorside, Belgium. A meat inspector, in order to show confidence in his own judgment, consumed some saveloys and died within 5 days of salmonellosis (*Salm. enteritidis*). It is variously related that his action was in refutation of a doubt as to the fitness of the carcass from which came the meat used in making the saveloys, or a doubt about the fitness of the saveloys themselves.

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Chapter VI

POSSIBLE SOURCES AND MODES OF INFECTION

TRACING the primary source of salmonella bacilli in food-poisoning outbreaks has always been found an exceedingly difficult problem. Although a considerable amount of information on the subject has been collected, recent knowledge attained by observers, after studying a large number of individual cases and outbreaks, has revealed that in many instances although the definite cause of the illness has been discovered, i.e. infection of the food consumed, by salmonella organisms, the primary source of the bacilli (the actual reservoir) and the mode of transmission, thence to the food ingested (path of infection), could not be ascertained, or only incomplete information was obtainable which eventually proved of little value to the investigators in discovering these important issues. This, perhaps, was due in a measure to delay in commencing the investigations or to difficulty in obtaining the necessary material for bacteriological examination. Now that food poisoning is notifiable and improved technical procedures are available for investigation, as well as increased laboratory facilities, doubtless this unnecessary delay will be avoided.

Before discussing the possible sources and modes of infection, it may be of interest to quote the remarks of Topley and Wilson (1946):

> Attention should be drawn to the apparent infrequency of Salmonella food poisoning in man in spite of the widespread incidence of infection in domestic animals, and their products used for food, and in rodents. Scott (Report 1929) explained this discrepancy on the assumption that large doses of *Salmonella* were probably required for infection of man. In typhoid, cholera and probably dysentery, minute amounts of infective material may suffice to cause disease, but in food poisoning, large doses are generally necessary. Savage (1942) had ably developed this theme in relation to paratyphoid fever; and Hormæshe, Peluffo and Aleppo (1936) have supported it by human volunteer experiments with *Salm. typhi-murium*. These workers found that adults might ingest as many as 2,000 to 4,000 million organisms without suffering from any symptoms at all, or from not more then mild afebrile diarrhoea. Infants and old persons, however, appear to be considerably more susceptible and fatal cases are restricted mainly to these two age groups.

The known ways in which food may become infected by salmonella organisms are as follows:

(*a*) Meat from diseased or infected animal or passive carrier.
(*b*) Milk from an infected animal.
(*c*) Salmonella infection transmitted by duck eggs.
(*d*) Salmonellosis in ducklings, ducks, chickens, and their eggs.
(*e*) Infection transmitted by rats or mice—rat viruses.
(*f*) Human carriers (infected sufferer or passive carrier).
(*g*) Flies and other insects as possible carriers of salmonella infections.

Meat from a Diseased or Infected Animal

In the early days, Bollinger maintained that the flesh of animals suffering from septic and pyaemic diseases was unfit for human consumption. There seems little doubt that animals, especially cattle and pigs, may suffer from umbilical infections, intestinal and other diseases caused by salmonella organisms. The disease is usually acute and often fatal, but the carcass need not necessarily be noticeably unhealthy.

In this country the majority of the reports on food poisoning outbreaks contain little information regarding the health of the animal from which the food is derived. This shows the importance of ante-mortem and post-mortem examinations of food animals.

Savage (1932) remarks:

> The only sources of infection of which we have accurate information are animals or birds either suffering from Salmonella infections or with the bacilli present in a carrier condition. In one group we can include animals used by man for food. Calves and cattle suffer from Salmonella infections, the strain being usually B. enteritidis, less commonly B. aertrycke, while B. morbificans bovis was originally isolated in 1893 from a diseased cow. Swine infections with Salmonella bacilli are well known, either as a cause of definite enteritis or as secondary invaders in swine fever.

Scott (1940) examined the mesenteric lymph nodes from 1,000 pigs. Thirty-eight strains of salmonella were isolated, an incidence of 3·8 per cent. The species isolated were *Salm. cholerae-suis*, *typhimurium*, *thompson*, *newport*, *enteritidis*, *london*, *dublin*, and *reading*.

Rubin, Scherago, and Weaver (1942) investigated 'The occurrence of Salmonella in the lymph glands of normal hogs with the following results:

> The mesenteric lymph glands from apparently normal hogs have been examined for the presence of Salmonella, using the tetrathionate meidum of Kauffmann for enrichment. Of 40 lots of hogs, consisting of

25 animals each, 19 (47·5 per cent) yielded Salmonella. Of 50 hogs examined individually, 5 (10 per cent) yielded Salmonella. The types of Salmonella which were isolated are as follows: S. typhimurium, S. choleraesuis var. kunzendorf, S. oregon, S. anatum, S. give, S. bareilly, S. derby, S. new brunswick, S. bredeney, S. enteritidis, S. lexington, S. newington, and S. worthington.

Clarenburg, Vink, and Huismann (1949) examined the mesenteric glands together with the faeces of 503 healthy pigs for the presence of salmonellae. In 14 animals (2·78 per cent) salmonellae were demonstrated in the mesenteric glands; the faeces gave negative results. The organisms isolated were: *Salm. typhi-murium*, *dublin*, *enteritidis*, *newport*, and *give*. See also Varela and Zozaya (1942), Edwards and Bruner (1943), Taylor, Macdonald, and Sivell (1951), Buttiaux, Gaumont, and Morel (1951), Smith and Buxton (1951).

Few instances of sheep being infected with salmonella organisms have been described. Fourie (1936) recorded the following outbreak which occurred in Uberuher, South Africa, in 1919. Of 4,000 inhabitants more than half were affected. The mutton which was responsible was obtained from sheep which had gastroenteritis. The carcasses were released for sale as, on inspection, nothing was found beyond slight reddening of the mucous membrane of the stomach and intestines. From the carcass and from the stools of infected humans *Salm. aertrycke* (*Salm. typhi-murium*) was isolated. Most cases occurred when food, which was not properly cooked, was consumed, and in some cases infection took place merely by handling infected meat. So that, in this case, the condition in the human beings was more of an infection than an intoxication.

Edwards (1947) isolated 6 types, including *Salm. typhi-murium*, *Salm. enteritidis*, *Salm. cholerae-suis*, and *Salm. paratyphi* B. from 33 outbreaks. See also Jesson (1950), Wall and Wall (1952), Déom and Mortelmans (1954).

In the annual report of the Chief Medical Officer, Ministry of Health for 1935, an outbreak occurred in Lancashire in which beef from the carcass of an animal suffering from salmonella septicaemia when slaughtered was responsible for 174 cases with 8 deaths. The carcass was not noticeably unhealthy, but from all parts of it examined *Salm. typhi-murium* (*Salm. aertrycke*) was isolated. In many of the cases the meat had been consumed in the form of pressed beef.

In a number of outbreaks due to the consumption of meat from a diseased animal (especially in Germany), it has been ascertained

afterwards to have been 'emergency-slaughtered'. According to German authorities, four-fifths of the outbreaks of meat poisoning are due to cattle slaughtered when on the point of death, suffering from some septic or diarrhoeal condition, slaughter having been effected under private or subsequently unascertainable conditions. In 61 large outbreaks of food poisoning between 1869 and 1898, affecting 5,000 persons with 76 deaths, the meat of cows was incriminated in 38, of calves 15, of oxen 3, of pigs 2, and of horses 2. Meyer (1929) recorded 120 outbreaks between the years 1923 and 1928 which were due to this cause.

Elkeles (1930) reviewed the subject and quoted figures from Standfusz which demonstrated that, of clinically recognizable infections, 22–47 per cent are characterized by gastro-intestinal symptoms, 11–21 per cent by septicaemia, 11–19 per cent by disturbances during parturition, and 19–48 per cent by other manifestations.

Doubtless under inadequately controlled conditions, opportunities may occur for the transference of infection from diseased animals to healthy meat where the emergency slaughter of sick animals takes place in the same room as healthy carcasses are being dressed. The careless handling of local lesions of a diseased animal may also be a possible source of salmonella infection. Ostertag has pointed out that as a result of the analysis of 85 recorded outbreaks of food poisoning during the years 1880–1900, most of which occurred in Germany,

> prove anew the especially dangerous character of the meat of calves affected with sepsis in association with umbilical affections and also of cows which have to be subjected to emergency-slaughter on account of inflammatory processes after parturition or on account of peculiar affections of the intestines and udder.

Savage (1920) investigated and recorded the following interesting and typical outbreak definitely connected with food from a diseased animal:

> On Friday, 8th May, 1908, in Murrow, a village in Cambridgeshire, a woman purchased some pork bones from a local butcher and that evening used them to make some brawn. The following morning the brawn was emptied out of the saucepan in which it had been made and, without cleansing the vessel, potatoes and asparagus were cooked in it. These vegetables were eaten for midday dinner by 4 persons, and all were subsequently attacked with vomiting, diarrhoea, and the other symptoms of food poisoning, two in the night and two next morning. The husband who was away at midday remained well and unaffected.
>
> On the Monday, two days later, the brawn made up into pork cheeses (a local name for brawn) was given away to three different

neighbours and was consumed by a further 14 persons, all of whom were attacked with similar symptoms, after an incubation period varying from 12 to 48 hours. Three of the 18 attacked died. No one eating the brawn escaped.

None of the brawn was available for examination, but from the only fatal case investigated a Gaertner group bacillus (Salm. aertrycke) was isolated, and its connection with the outbreak was further proved by the fact that it was agglutinated in high dilution by the serum of three survivors.

The brawn was home prepared, and the materials were slowly heated for several hours with a short boil at the finish, but obviously actual boiling temperature was not reached. That the Gaertner bacilli were present before preparation and survived cooking is evident from the infection imparted to the vegetables through the uncleansed saucepan. Further inquiries elicited that the pig which supplied the bones for the brawn had suffered from local injury or disease of one leg, no doubt due to infection by this food-poisoning bacillus.

Salm. typhi-murium (*Salm. aertrycke*) and *Salm. enteritidis* have been isolated in recent years from sick animals by several observers, including Gheorghiu and Costin (1927), Lachenschmid (1931), Edwards (1934), Lovell and Hughes (1935), Hohn and Herrmann (1935), and Ferrario (1935).

Cherry, Bailey, Scherago, and Weaver (1943) carried out investigations into the occurrence of salmonella in retail meat products. They state that

samples of various types of meat products were obtained from retail markets and examined for the presence of Salmonella, using the tetrathionate enrichment method of Kauffmann and the selenite enrichment method of Leifson. Of the 250 samples analyzed 13 (5·2 per cent) were found to contain Salmonella. The incidence was found to be greater in pork products than in beef. The following types of Salmonella were isolated: S. typhimurium, S. give, S. derby, S. anatum, S. newport, S. bredeney, S. senftenberg, and S. newington. Evidence is presented to indicate that the most probable source of the Salmonella is the animals from which the meats were obtained.

A large outbreak of food poisoning caused by *Salm. typhi-murium* probably due to the consumption of meat from a compulsorily slaughtered animal was recorded by Ahrens (1942) and described by Savage (1943):

The author describes an outbreak of food poisoning with the usual symptoms during the first week of October, the majority of the cases developing on October 1–4. There were about 270 cases, duration for most part 3–5 days; no deaths. The incubation periods were, generally, 4–6 hours. The vehicles of infection were sausage (Knackwurst) and chopped meat, all from the same source. *Salm. typhi-murium* was isolated from this material and also from the great majority of the

numerous stools of patients examined. The author agrees that it is not possible to determine with certainty if it was an original infection of the meat or a secondary infection. On the grounds that the slaughtered animal was not in good condition and that from the rest of the meat there were five cases of gastro-enteritis, he concludes that it was probably due to an intravital infection of a compulsorily slaughtered animal.

DISTRIBUTION OF SALMONELLA IN MEATS

Animal	*Meat*	*No. of Samples*	*Per cent Positive*	*Salmonella Isolated*
Pig	Brains	10	10	*S. derby*
Pig	Chops	21	9·5	*S. derby* *S. senftenberg*
Pig	Ham	17	0	
Pig	Kidney	10	0	
Pig	Liver	30	20	*S. anatum* (2) *S. bredeney, S. give* *S. newington* *S. newport, S. typhi-murium*
Pig	Sausage (fresh)	44	2·3	*S. typhi-murium*
Pig	Sausage (smoked)	5	0	
Pig	Other meats	33	0	
Ox	Hamburger steak*	24	8·3	*S. senftenberg* *S. typhi-murium*
Ox	Liver	14	0	
Ox	Sirloin	3	33·3	*S. senftenberg*
Ox	Other meats	23	0	
Sheep	Chops, Fries	11	0	
Chicken	Liver	3	0	
Pork and Beef	Loaf	2	0	
		250	5·2	

*Some samples probably contained both beef and pork.

Scott (1940) draws attention to the salmonella organisms—*Salm. typhi-murium*, *thompson*, *newport*, and *cholerae-suis*, isolated from the spleens and mesenteric glands or spleens of about 5 per cent of apparently healthy pigs slaughtered in this country.

Savage (1956), in discussing more complete ascertainment of the animal reservoir of salmonellae, points out:

> We know much about salmonellosis in animals, but are still very incompletely informed about their quantitative distribution. To extend our knowledge of reservoirs of salmonella strains we need more extensive surveys of animals that may possibly be associated with food-poisoning. A good illustration is *Salm. dublin* infections in cattle. We know of a number of scattered pockets of infection, but we do not know its

extent in cattle generally or if these infections are on the increase. Recent evidence that salmonella-infected animal foods may spread the disease to healthy animals is a new light on a possible method of spread. Combined veterinary and bacteriological studies could contribute much to our knowledge of salmonella reservoirs.

Grütter (1944) records that after *Salm. enteritidis* had been isolated from the meat of a slaughtered calf, 7 heifers of the 15 cattle on the same farm were found to be permanent excretors of this micro-organism. They were slaughtered and it is stated that in each the hepatic parenchyma was affected with different bacteria. All 7 heifers had liver fluke infestation and it is suggested that the bacterial infection resulted from this parasitic infestation. Grütter suggests that liver fluke predisposes to secondary bacterial infection more commonly than is usually realized and that in districts of heavy fascioliasis, special attention should be paid to the possibility that bacteria may be excreted by affected cattle.

Surveys have been made from time to time on the incidence of salmonella in cattle and pigs but apparently little recorded information has been available on the occurrence of any of these organisms in carcasses of animals passed for human consumption. Members of the Salmonella Sub-Committee of the Public Health Laboratory Service carried out a survey during the four years 1950–3, published in 1955. Samples were collected from the carcasses of cattle, calves and pigs at time of slaughter from six abattoirs, at Birkenhead, Birmingham, Bradford, Newcastle, Nottingham, and Taunton. The material was cultured and bacteriologically examined for salmonellae. The samples consisted of peritoneal swabs, livers, spleens, and mesenteric glands, bile, and faeces. The results are as follows. In cattle, *Salm. dublin* was the commonest type isolated, comprising 42 out of a total of 450 strains. Three strains of *Salm. typhi-murium* were also isolated. The isolations were from a few specimens of bile, liver, spleen, mesenteric glands, and faeces, but negative results were obtained from swabbing the peritoneal surfaces of 1,518 sides of beef. No salmonellae were isolated from the livers and spleens of 494 calves. The investigators remark:

> The negative results obtained from 1,518 peritoneal swabs suggest that the risk of beef being contaminated at the abattoir is small, but a number of isolations of *Salm. dublin* from bile and livers, show that a small proportion of cattle reaching the abattoir are infected.

As *Salm. dublin* is much the commonest Salmonella to be found in cattle in this country, infection of cattle products with Salmonellae of

types other than *Salm. dublin* is more likely to be due to contamination after slaughter than to infection of the animal during life.

In pigs, *Salm. dublin* was isolated at the Newcastle laboratory from 6 out of 452 samples of bile and from the liver, spleen and glands of 1 out of 27 pigs. This Salmonella was not isolated by the other laboratories, which obtained negative results from 500 samples of bile, 1,130 samples of liver, 687 of spleen and 677 faeces. Two strains of *Salm. typhi-murium* and one of *Salm. anatum* were isolated from 1,146 peritoneal swabs, and one strain of *Salm. typhi-murium* was isolated from 409 samples of mesenteric glands.

Animal Carriers

Buxton (1957) states:

> The origin of many Salmonella infections in man and animals is the infected carrier animal or food products derived from such an animal. Individuals which successfully combat infection may remain symptomless carriers for varying periods, during which their faeces will be infected intermittently with Salmonella. The majority of such animals develop no clinical signs of infection. They form the main sources of infection for other animals and are most difficult to detect by bacteriological or serological methods. The extent to which different species act as carriers or reservoirs in infection varies, and our knowledge of the relative importance of each species depends upon the extent and the number of surveys which have been made, the types of media employed and the suitability of the material examined. Poultry and pigs apparently constitute the greatest reservoir for Salmonella.

Milk from an Infected Animal

Milk-borne outbreaks are usually of an explosive nature and widespread. Milk may be the vehicle of salmonella strains or staphylococcal toxigenic organisms. The source of infection usually comes under one of the following headings: (1) infection from a diseased cow; (2) through the cow, but caused by human contamination; (3) contamination of the milk after leaving the cow.

There are a number of recorded outbreaks where salmonella organisms have been isolated from the milk of a diseased cow and/or from the faeces. Minett (1938) isolated 38 strains of *Staphylococcus aureus* from udders of cows. Seven had acute mastitis, 8 with chronic mastitis and the remainder from cows with normal udders. See also Kinloch, Smith, and Taylor (1926), McAllan and Howie (1931), Savage (1932), Tullock (1939).

Conybeare and Thornton (1938) investigated a large outbreak (130 cases) which occurred in a town in Wiltshire, chiefly among

school children. *Salm. enteritidis dublin* was isolated from the milk and faeces of one of the cows. The mode of transference from the animal reservoir to the human subject was clearly established.

In 1954 Norton and Armstrong recorded a milk-borne outbreak (252 cases) which occurred in County Durham districts due to raw tuberculin-tested milk infected with *Salm. typhi-murium*. The organism was isolated from the milk of a cow and her faeces, from 196 samples of faeces from notified cases, and from 15 out of 23 milk handlers. This is apparently the only milk-borne outbreak recorded for this particular organism and isolated from an animal and its faeces.

Salmonella Infection Transmitted by Duck Eggs

The infection of ducks and their eggs by salmonella organisms such as *Salm. typhi-murium* or *Salm. enteritidis* is not uncommon. Much research work on this subject has been carried out. It would appear, however, that only certain breeds of ducks are susceptible in infection. It has been suggested that as ducks are naturally dirty feeders, they may in their wanderings pick up and swallow infected material and so transmit the living organisms to the egg yolk. Or the pathogenic organisms may pass through the porous egg shell from an outside source. A person purchasing a setting of such infected duck eggs may breed infected ducklings—a possibility which cannot be overlooked.

The importance of the duck's egg as conveying salmonella infection was brought into prominence by Lecoq (1906) and Scott (1930, 1932, 1933). See also Clarenburg and Dornickx (1932), Lovell (1932), Beller and Reinhard (1934), Miessner and Kofer (1934), Seligmann (1935), Hohn and Herrmann (1935), De Koning (1936), Jansen (1936), Kathe and Lerche (1936), Brown, Coombs, and Wright (1940), Garside and Gordon (1940); Gordon and Buxton (1945), Garrod and McIlroy (1949), Lawrence Smith (1950), Eedy (1950), Blaxland and Blowers (1951), Miller (1952).

In 1932, Dalling and Warrack stated:

> So far we know bacilli of the Salmonella group have not been previously obtained from the interior of duck eggs. Some eighteen months ago we heard that a large number of ducklings had died on a farm. We asked for blood from the parent ducks for agglutination test. A number of the samples agglutinated B. gartner. We obtained 12 ducks—6 positive by agglutination and 6 negative—and kept them at the laboratories. Repeated tests were made during the twelve months. The negative reactors remained negative and the positive remained

positive. One hundred and sixty-six eggs were laid by the positive reactors; from 7 of these eggs laid in clean bedding B. gartner was obtained. The 5 ducks were then killed and B. gartner was found in the ovary in each instance but in no other organs.

Recently D. Wm. Scott enabled us to get 18 ducks whose serum agglutinated B. aertrycke. From the eggs of some of these positive reactors Scott obtained B. aertrycke. This flock has been under observation for about a month. The negative reactors remain negative and the positive still react. There is, therefore, some hope that one may be able to clean an infected flock by eliminating the positive reactions under subsequent observations. We have tested the blood from 14 flocks, including 1,231 ducks. Positive reactors to either gartner or aertrycke have been found in 8 of these flocks.

Scott (1932) remarks:

Three recent outbreaks of acute gastro-enteritis due to aertrycke-infested eggs were described. In each a single case occurred in a family—one fatal; in each the aertrycke bacillus was isolated from the human excreta (or internal organs); in each the infection was imputed to the consumption of a duck's egg (two fried, one raw), and in each this suspicion was confirmed by the discovery of aertrycke-infected eggs from the corresponding flock of ducks. No connection between the three outbreaks could be traced, though two were near each other. In one flock all the ducks (9) were found infected, B. aertrycke being present in spleen, ovary and intestinal contents and in an egg removed from the oviduct. In another flock 18 out of 46 showed serological evidence of aertrycke infection and at least 4 of these laid aertrycke-infected eggs. In the third flock 2 of 5 showed serological evidence of aertrycke-infection and at least 1 laid aertrycke-infected eggs.

The importance of suspecting an egg as the vehicle of infection in solitary cases of food poisoning was emphasised, as such cases may be otherwise inexplicable.

In July 1949 over 100 of the staff and patients at a hospital in London were affected by an outbreak of food poisoning. *Salm. typhi-murium* was isolated from a number of those attacked. There is little doubt that the vehicle of infection was the 'queen pudding' which was eaten by some 600 persons. This pudding was made in two stages. First, breadcrumbs were mixed with a custard made with milk and the yolks of 200 duck eggs and baked in the oven for about half an hour. The whites of the eggs were then beaten up with sugar and poured on to the top of the sponge and grilled until the surface was slightly browned.

No members of the kitchen staff suffered from suspicious illness before the outbreak and it can be assumed that infection originated from the duck eggs. It is possible that parts of the sponge which had been baked for half an hour may not have reached sterilizing temperatures, but it is certain that the heat treatment

given to the white of the eggs was not sufficient to kill the organisms on this portion of the pudding.

This outbreak is a salutary reminder of the potential danger of consuming duck eggs without adequate heat treatment. Furthermore, unless the origin of the eggs is known and it can be assured they are not infected with *Salm. typhi-murium*, it is dangerous to use duck eggs in the preparation of meringue (Medical Officer, 1949).

Eedy (1950) mentions a case of food poisoning caused by the consumption of a duck egg. *Salm. typhi-murium* of vi-phage type 4 which was isolated from the urine and faeces of the patient and from 8 out of 47 eggs from the incriminated flock of ducks, but not from rectal swabs from the birds, with the exception of the drake. Bacteriological examination of the eggs 3 weeks after elimination of the drake gave negative results.

Smith and Buxton (1951) examined faeces from 500 ducks and found 1·2 per cent positive.

Salmonellosis in Ducklings, Ducks, Chickens and their Eggs

Garside and Gordon (1940) record instances of salmonella infections in ducklings. They state that in course of routine laboratory work infection with *Salm. typhi-murium* and *Salm. enteritidis gaertner* has been diagnosed on many occasions. From 1933 to 1939 *Salm. typhi-murium* was isolated in 57 instances and *Salm. enteritidis gaertner* in 8, from chicks, ducklings, pheasant chicks, turkey poults, goslings, and pigeons. It appears that ducklings up to 4 weeks of age were most susceptible to natural infection.

They mention an epidemic recorded by Rettger and Scoville (1920) in America, a disease termed 'Keel', which caused losses amongst thousands of ducklings, approximating 90 per cent of those hatched. A salmonella organism was isolated from the affected ducklings and named by them *Salm. anatum*.

Later, Edwards and Rettger (1927) re-examined the strains isolated from the ducklings and isolated 2 types, 1 identical with *Salm. typhi-murium* and the other a new species for which the name *Salm. anatum* was retained.

In this country Doyle (1927) recorded an outbreak in young chicks with a mortality of 100 per cent from which *Salm. typhi-murium* was isolated.

Gaiger and Davies (1930) investigated a severe epidemic of 'Keel' disease involving a loss of 4,000 ducklings during 1 year. The infective agent was a salmonella organism identical with *Salm. anatum* described by Rettger and Scoville (1920); later, however,

Hall (1932) and Lovell (1932) re-examined the organisms and came to the conclusion it was *Salm. enteritidis gaertner*. 'Keel' disease in ducklings was described by Dunning (1939) in an outbreak in South Africa, in which an organism closely resembling *Salm. enteritidis gaertner* was found.

Garside and Gordon, in their conclusion and summary, state:

> In this, as in other outbreaks of Salmonella infection observed, vermin (rats and mice) seem to play an important part in appearing to act as natural reservoirs and disseminators of infection.

Scott (1933) suggested that the eggs are probably infected during their formation in the oviduct, but the bacilli may gain access through the intact shell. In his paper on the subject (1930), he mentions several outbreaks, including a typical one which occurred at Darlington in 1927. A trifle was consumed by 10 out of a party of 12 persons. All the 10 were seriously ill, owing to an aertrycke infection, while the two who had no trifle but had shared in all the other food, remained well. The cream of the trifle had been prepared by whipping the whites of ducks' eggs.

Beller and Reinhard (1934), who examined 1,500 ducks' eggs from 34 farms in Germany, found that about 1 per cent contained salmonella organisms.

De Koning (1936) described an outbreak of food poisoning in 60 people from the consumption of ice cream containing raw duck eggs. Twenty-two of the 25 ducks which produced the eggs reacted positively to the agglutination test for *Salm. typhi-murium*, and in studying the same outbreak Jansen (1936) isolated identical strains of *Salm. typhi-murium* from the patient's faeces, from the yolks of eggs, and from the degenerate ovaries of the reactor ducks.

Hedstrom (1941) reported an outbreak of *Salm. typhi-murium* infection in fowls, turkeys, and geese on the same farm, which occurred simultaneously with an illness in the owner's family in which identical strains of the organism were recovered.

In 1941 Müller recorded 23 cases of food poisoning admitted to a Hamburg hospital, each with the history of having eaten raw duck eggs. *Salm. typhi-murium* was isolated from 18 of the cases and *Salm. enteritidis* from 2.

In the investigations carried out by Gordon and Garside (1944) they remarked that the most striking feature of the cultural work was the frequency with which the organisms were recovered from the ovary, continuing:

> It was found, however, that out of 45 infected ovaries only 5 were active, and consequently potential sources of transmission at the time

of examination. Although the remaining birds from which the organisms were recovered from the ovary must be considered as potential transmitters when and if the ovary was functioning, this was not borne out by our negative breeding experiments or by the negative results obtained on the cultivation of over 2,000 eggs. . . . In connection also with egg transmission, it is of interest to note that in the overwhelming majority of reported cases of food poisoning of human beings in which ducks' eggs are suspect, the recovered organism is *S. typhi-murium* and not *S. enteritidis*. Although they are not altogether conclusive, all our experimental findings bear out our previous contention, which was based on field observations (Garside and Gordon, 1940) that in Salmonellosis in duck egg transmission, if it does occur, plays a minor role in the dissemination of infection.

The following interesting case of food poisoning in man, probably caused by the consumption of a duck egg, is recorded by Gordon and Buxton (1945):

On 23rd May, 1944, a man aged 49 was admitted to Rotherham Municipal Hospital suffering from acute gastro-enteritis. Apparently symptoms first appeared on 21st May, a few hours after eating a duck egg fried with ham for breakfast. *Bact. typhi-murium* was isolated from the blood and faeces of the patient, who subsequently died on 27th May.

The duck egg was obtained from a neighbour on 21st May, and had been picked up that morning, although the time of laying is unknown. The egg appeared quite normal. The premises where the ducks were kept adjoined a railway line on one side, and a canal on the other, and were also used for keeping pigs, fowls, horses, rabbits and goats. Four ducks and a drake were kept and there had been no recent deaths in the ducks or fowls. The duck eggs had been eaten regularly by the family as well as by neighbours and their children, and there had been no complaints of any description during recent months. The premises were in a most unsanitary condition, consisting of crude wooden buildings with poor floors and no proper drainage. Rats were prevalent, but no attempt had been made to exterminate them by the use of poisons. No mice were seen.

[Sixteen of the duck eggs were examined. These were incubated at 37°C. for a period of 5–7 days before examination.]

Technique.—The eggs were placed in a wooden holder and a few drops of methylated spirit were poured over the shell in the region of the air space. The spirit was ignited and allowed to burn off and a hole bored in the shell with a dentist's electric drill which had been previously sterilised in methylated spirit. A sterile Pasteur pipette was inserted through the hole and approximately 1 ml. of yolk was withdrawn and inoculated into 5 ml. of tetrathionate broth. In this way it was possible to obtain yolk without its coming in contact with the egg white or the shell. The rest of the contents of each egg was broken and poured through a sterile funnel into a flask containing 100 ml. of tetrathionate broth. The shell was then ground up with fine sterile sand and placed in another flask containing 100 ml. of tetrathionate broth. The cultures were incubated at 37°C. for 18–24 hours and a loopful plated on to Mac-

Conkey's agar and on to brilliant green agar (1/75,000). Suspicious colonies were picked off and sown into lactose, maltose, dulcitol, 10 ml. of beef infusion broth, and on to agar slant. Organisms which failed to ferment lactose but fermented maltose and dulcitol with gas production were then tested by the rapid slide agglutination method with a number of the Salmonella sera supplied by the Oxford Standards Laboratory. To complete the identification of the organisms, agglutinations to titre were carried out using both *Bact. typhi-murium* O and H specific sera.

[Results] Three of the 16 eggs examined yielded *Bact. typhi-murium.*

1. Egg laid by Duck 1150 on 23rd June: *Sal. typhi-murium* was isolated from all three cultures, i.e. from yolk, yolk and white, and from the ground-up shell.

2. Egg laid by Duck 1147 on 23rd June: *Sal. typhi-murium* was isolated from the shell only.

3. Egg laid by Duck 1147 on 11th July: *Sal. typhi-murium* was isolated from yolk, and yolk and white, but not from the shell.

The titres of these birds at the tests previous to the laying of the infected eggs are of interest. Ducks 1150 and 1147 gave completely negative reactions in the dilutions used, the day before eggs 1 and 2 were laid; while 1147 again gave a negative agglutination seven days before laying the other infected egg.

Post-mortem and bacteriological examinations of ducks.

Cultures were made from the liver, heart, gall bladder and spleen, and sown into peptone broth tubes and on MacConkey agar plates. The entire ovaries were ground up and placed in 100 ml. quantities of tetrathionate broth, while the contents of the intestinal tracts were squeezed into similar flasks of tetrathionate broth, and after 24 hours' incubation at 37°C. were plated on MacConkey's agar and 1/75,000 brilliant green agar. The isolation and identification of *Sal. typhi-murium* was then carried out as described for the egg.

[Results] *Sal. typhi-murium* was isolated from the ovaries of Ducks 1147 and 1150 and from the intestinal tract of Duck 1149.

[Gordon and Buxton continue] The evidence recorded in the present paper appears to be more convincing than any so far produced in this country, incriminating the duck egg as a source of gastro-enteritis in man. Not only were reactors to *Sal. typhi-murium* found in the flock from which the suspected egg was obtained, but the same organism was isolated from the yolk and shell of 3 of the 16 eggs laid by the four ducks, from the ovaries of both ducks which laid the infected eggs, and from the intestinal tract of a third duck.

[Summary] *Sal. typhi-murium* was isolated from the blood and faeces of a patient who died of gastro-enteritis following the consumption of a fried duck egg. A fortnight later, agglutinins to *Sal. typhi-murium* were found in the sera of three of the four ducks composing the flock from which the egg was obtained; the serum of the fourth duck became positive after a further four months. *Sal. typhi-murium* was cultured from the yolk and shell of 3 of the 16 eggs laid by the four ducks, and the same organism was isolated from the ovary of the two ducks which laid the infected eggs, as well as from the intestinal tract of a third duck.

Gillespie (1946) records a further report on the above case as follows:

> Salm. typhi-murium vi-phage type 2 was isolated from the blood and faeces of a patient dying of acute gastro-enteritis following the consumption of a duck's egg. The same vi-phage type strain was isolated from some of the eggs, the ovaries and intestinal tract of the ducks composing the flock from which the egg was obtained.

Blyth Brooke, Douglas, and Taylor (1950) reported a severe case of gastro-enteritis due to *Salm. typhi-murium* in a boy aged 2 years. Infection was possibly conveyed by eggs from a sick hen kept with two others in a yard at the boy's home. *Salm. typhi-murium* was isolated from a blood clot from this hen and from caecal contents after death. *Salm. thompson* was also isolated from the blood clot of this bird and from one of the others. No salmonellae were obtained from the eggs of any of the hens. The investigators suggest 'that the possible role of back-yard poultry in the spread of salmonella infections is worthy of further investigation'.

Bernstein (1952) made an investigation designed to provide information on the rate of infection with salmonella of marketable hen eggs in England during the period June 1950–May 1951. The contents of 3,648 eggs from various sources at home and abroad were examined, but no members of the salmonella group were isolated.

Jellard (1956) records an explosive severe outbreak of food poisoning which occurred at a preparatory school. The meal, which was partaken of by 47 boys and 10 staff, consisted of hot roast beef and vegetables and a cold egg custard pudding. The pudding, which was partly prepared the previous day, contained the yolks of several dozen eggs mixed with gelatine powder and sugar and lightly cooked. It was afterwards placed in a large bowl (about three-quarters full) and left in a cellar overnight. Shortly before lunch the following day the whites of the eggs were beaten up with sugar and added to the yolks and gelatine mixture and preparation of the pudding completed. The gelatine was from a batch which had been in use for some time. Between 11 and 48 hours after the meal, 56 of the boys and staff, including the cook, were taken acutely ill, attacked with abdominal pain, diarrhoea, vomiting, and fever which lasted from 1½ to 3 days, and in some cases diarrhoea persisted for some days. All the patients recovered. Subsequent bacteriological examination of the faeces revealed that 18 of the 47 boys attacked were excretors of *Salm. typhi-murium.*

Infection during preparation of the custard could be excluded for two reasons: (1) the cook who prepared the food was affected with the others who consumed it and was found on examination not to be an excretor, and (2) contamination of the egg-gelatine mixture by mice was impossible owing to the shape of the bowl in which it was stored.

The crate in which the eggs were packed was examined and it was found that the bottom, on which a layer of eggs had rested, consisted of an unsavoury many-layered sandwich of decaying egg, fly maggots, and mouse droppings. Other crates examined also contained mouse excreta. Contamination of these containers with *Salm. typhi-murium* could easily have occurred from excreta by mice at the egg packing station. Enquiries revealed that it was not unusual to find mice in the crates.

Regarding the custard pudding, the high rate of attack (98 per cent) suggests that the organism had multiplied abundantly during the slow cooling over-night of the partly cooked food. It has been stressed from time to time by medical authorities of the importance that 'made up food' be consumed on the day that it is prepared and that immediately after preparation it be placed in a refrigerator and not left exposed to room temperature. It would appear essential that all crates and boxes used for the packing and transport of eggs be examined periodically and thoroughly cleansed before being used again.

Edwards, Bruner, and Moran (1948) recorded that in the United States, fowls were the greatest reservoir of salmonellae. A high incidence of a given type in fowls in any area was accompanied by a high incidence of the same type in man.

Gaumont (1950) found that salmonella infection was fairly prevalent among chickens in Northern France. He examined 245 birds, or groups of birds, and isolated 41 salmonellae (16·7 per cent.)

Salm. typhi-murium infection has been recorded as occurring in canaries and parrots, Beaudette (1926); geese, Baars (1931); pigeons, Schutt (1951), Sorum (1953); turkeys, Marek, Meuszynski, and Larski (1953); elephants and chimpanzees, McGaughey, Schmidt, Velandapillai, and Weinman (1953).

Infection Transmitted by Rats and Mice

Rats and mice are known to be susceptible to infection by salmonella. The rat is probably the host of *Salm. enteritidis* and the mouse harbours *Salm. typhi-murium*, and they may, when so

infected or in the carrier state, excrete the organisms in their faeces and urine for considerable periods. Food prepared and left exposed in insanitary premises attracts rats and mice and is liable to be so infected.

Savage and Read (1913) examined the internal organs and intestinal contents of 41 rats and were able to isolate *Salm. enteritidis* from the spleen of 5 rats. No member of this group was isolated from the intestinal content. This proved that while rats may be infected with organisms of the salmonella group, these bacilli are not normal inhabitants of their intestinal tracts.

Savage and Bruce White (1923) examined 96 rats caught in two slaughter-houses. They isolated *Salm. enteritidis* from 6 of them; 3 of them harboured the organism in their intestines, thus demonstrating that the infection of meat from this source is possible. Meyer and Matsumura (1927) made an examination of 775 wild rats from the district of San Francisco and found 58 harboured *Salm. enteritidis* (28) or *Salm. typhi-murium* (30).

SALMONELLA INFECTIONS OF LIVERPOOL RATS, 1936

Rats Examined	*Number Positive*	*Percentage Positive*	*Rats Examined*	*Number Positive*	*Percentage Positive*	*Rats Examined*	*Number Positive*	*Percentage Positive*
250 Jan. to Mar.	44	17·6	250 Apr. to June	10	4·0	250 July to Aug.	—	0·4
	26 *aertrycke*			1 *aertrycke*			1 *enteritidis*	
175 City 75 Port	16 *enteritidis*		175 City 75 Port	7 *enteritidis*		175 City 75 Port		
	2 *newport*			1 *newport*				
				1 *thompson*				

The annual report of the Chief Medical Officer of the Ministry of Health for 1936 records an investigation of the salmonella infections of rats by Khalil, which 'illustrates the importance of the animal reservoir in which the various Salmonella types maintain their existence' (see accompanying table) and continues:

It will be seen that several of the types common in food poisoning can be found in the rat in much the same order of frequency; the enteritidis type (Gaertner), however, appears more commonly in the rat than it does in man, and there is reason to believe that it is the rat

type in particular, just as the Dublin type is bovine. As regards the other types, individual rats may become infected by them, probably by eating infected food, but, except in the case of aertrycke infection, rat epizootics apparently do not result from them.

As regards outbreaks, Jones and Wright (1936) record the case of a child who died from eating infected dried milk which was kept in an uncovered container. Mouse faeces were found in the food, and from these, as well as from the powder, *Salm. typhi-murium* was isolated. From the intestines of mice caught in the house within the next few days the same organism was cultivated.

Jordan (1929) stated that the widespread distribution of *Salm. enteritidis* in rats and mice might be of considerable epidemological importance. He reported that rats caught in various parts of Chicago frequently yielded this organism.

Savage (1932) says:

The fact that specifically infected rats and mice are vehicles of infection in food poisoning may be taken as established, and the infrequency of actual proof is probably due to the difficulties of the quest and a good deal to the failure of those responsible for collecting material, etc., to grasp the importance of this line of enquiry.

In America Welch, Ostrolenk, and Bartram (1941) carried out a number of investigations into the 'Role of Rats in the Spread of Food Poisoning Bacteria of the Salmonella Group'. In their summary they stated:

Excreta of rats naturally infected with Salmonella enteritidis held at room temperature may contain living organisms for at least 148 days.

Infection of rats and mice with very few organisms is possible when a virulent strain of Salmonella enteritidis is fed them by stomach tube.

Transfer of infection from an infected animal to cage mates has been carried through 7 colonies with rats and through 3 colonies with mice.

A study of rat and mouse excreta collected in areas throughout the United States indicates that only a small percentage (1·2 per cent) of these animals are excreting food poisoning organisms of the Salmonella type.

Topley and Wilson (1946) report:

The studies of Topley and his colleagues in this country and of Webster and his colleagues in the United States have shown that infection of rodents with *Salmonella* may result in an acute rapidly fatal septicaemia, a subacute enteritis which may or may not prove fatal, a chronic disease accompanied by changes in the liver and spleen, or by a completely symptomless infection.

Ludlam (1954) investigated the presence of salmonellae in wild rats in the Nottingham area. Salmonellae were isolated from

4·4 per cent of 518 rats killed in various types of premises during 1949–54. In addition up to September 1953, salmonellae were isolated from 6·4 per cent of 94 rats from a butcher's by-products factory. In the last 3 months of 1953 the incidence rose to 40 per cent of the 60 rats examined, and in the first 4 months of 1954 it was 27·6 per cent of 29 rats. Only two types of salmonellae were isolated from rats killed outside the factory, *Salm. enteritidis* var. *danysz*, and *Salm. newport*. Thirteen different varieties were isolated from rats caught in the factory and drains, the commonest being *Salm. enteritidis* bar. *danysz* and *Salm. typhi-murium*. 'The high incidence of Salmonellae in the factory occurred in a dense rat population and coincided with the over-loading of the factory with offal from autumn killings.'

Rat Viruses

Cultures of bacteria are sold frequently for the extermination of rats and mice. The Chief Medical Officer of the Ministry of Health, in his annual report for 1932, issued a warning regarding the use of virus preparations. He said:

> In 1929, and again in 1931, I called attention to the danger to human beings involved in the use of 'virus' preparations for the destruction of rodents and to the great caution necessary in employing them in circumstances in which contamination of food or drink might occur, either with the virus material itself or by the excreta of rats and mice infected with it. The occasion for this repetition was the outbreak of 1st November, 1931, in which 38 cases of food poisoning and 1 death was traced to the use of such a virus in a bake-house in Wigan.
>
> The vehicle was stuffed cow's heart or stuffed roast pork, the stuffing being the only part actually infected, and 2 patients consuming the stuffing alone. The stuffing when first made was not infected, as portions taken at once to a branch shop and part sold on 3rd November were harmless. The part which became infected remained on the shop counter overnight in a glass vessel placed on a bench in a passage at the rear of the shop. Next day it was mixed with the rest of the stuffing and this was poisonous in every case. On 26th October a mouse destruction material had been used on the premises. From remains of a tin of this bacterial material, from mice trapped on the premises, from the organs of the patient who died, and from the excreta of other sufferers Dr. Scott isolated bacilli which were identical in every particular with one another and with B. enteritidis (Savage, 1932).

A number of outbreaks have been recorded in which infection has been traced to the use of virus preparations for the extermination of rats and mice by Shibayama (1907), Jordan (1930), Boeker and Kauffman (1930), Kristensen and Bojlén (1931), Savage

(1932), Scott (1933), Tanner (1933), Harhoff (1941), Leslie (1942), Dathan, McCall, Orr-Ewing, and Taylor (1947).

Pleydell (1949) describes an outbreak of food poisoning in Birmingham among 14 persons after the consumption of cold beef sandwiches in a restaurant. The incubation period varied from 16 to 36 hours. *Salm. bovis morbificans* was isolated from the faeces of 12 persons. Examination of specimens from 8 members of the staff at the restaurant showed that 4 were secreting the organism; 2 had not been ill, but the other 2 had symptoms of enteritis. Bacteriological examination of rat droppings from the restaurant also revealed the presence of the above organism. Bacteriological examination of the rat poison which was being used in the restaurant showed, however, the presence of *Salm. enteritidis* (Gaertner). Pleydell states:

> These findings indicate that in all probability there was an epidemic of gastro-enteritis due to *Salm. bovis morbificans* affecting the rodent population in the vicinity of the restaurant and that unprotected food was contaminated by the infected rodents. The beef had been cooked and served hot for lunch and then left on an open dish in the basement kitchen for eight hours before being served as sandwiches. It is presumed that the contamination occurred during this interval.

Human Carriers

Almost every clinical case of human salmonellosis harbours and excretes the causal organisms in the faeces for an indefinite period after the illness as a 'temporary carrier' but on rare occasions for a prolonged period, as a 'chronic carrier'. There is little evidence that salmonella organisms are able to live and multiply in the human intestine and they usually disappear soon after recovery. The problem is a difficult one but proved cases of infection from human sources are relatively few. Instances have been recorded, however, when the organisms have been isolated from the faeces of persons not suffering from any food infection, i.e. symptomless, but infected persons. These 'healthy carriers' are a danger and certainly represent a hidden reservoir of potential food-borne infection.

In 1932 Savage expressed the view that the human carrier plays quite an insignificant part in the causation of food poisoning. In America, however, Jordan (1917), Geiger (1923), and Dolman (1943) attach importance to this source of infection.

Among food-poisoning outbreaks ascribed to human carriers was one which occurred in France in 1917, involving some 1,060

cases. It was investigated by Perry and Tidy (1919) and was proved to be due to *Salm. aertrycke* (*Salm. typhi-murium*). One case remained positive for 14 weeks.

Topley and Wilson (1936) remark:

That chronic Salmonella carriers, analogous to chronic typhoid carriers, are uncommon, there seems to be little doubt; but considering the frequency of rodent typhoid, it would be surprising if sporadic infections of human beings, particularly those used to handling food, did not occur fairly often. . . . Temporary carriers of this type must always be a danger to the human population. Their detection by bacteriological methods is bound to be difficult, since their carrier condition will often have cleared up before suspicion is cast upon them.

Stone (1942), who carried out in the Panama Canal zone research on the presence of 'Food Handlers in the Army and their relationship to "Salmonella Food Poisoning",' states that:

Sixty-six hundred and seventeen stool specimens were examined from approximately 2,000 individuals. Of this group, 49 were found to be carriers of intestinal pathogenic bacteria, or an average incidence of 2·46 per cent. Of this group 40 were carriers of Salmonella other than *Salmonella typhi*, 4 of *Salmonella typhi*, and 5 of Shigella. The Shigella carriers were made up of 3 positive for the Sonne bacillus, and 2 for the Flexner group of dysentery bacilli. Fourteen species of Salmonella were isolated . . . the findings on all carriers were confirmed by additional cultures after the first isolation. Some of these Salmonella carriers yielded positive cultures over a 60-day period. . . . Positive food handlers were generally asymptomatic and were associated with small epidemic outbreaks of food poisoning and diarrhoea.

Dolman (1943), in discussing human salmonella carriers, states:

Use of the newer selective media available for bacteriological examination of faeces from food handlers, such as brilliant green tetrathionate broth, bismuth sulphite agar, SS agar, desoxycholate-citrate agar, and Hynes' medium, is revealing a higher incidence of Salmonella carriers than was formerly suspected. Soon after the Provincial Laboratories in Vancouver began to carry out routine stool examinations of food handlers in the Armed Forces, three carriers of S. typhi-murium were identified in one regiment alone, none of whom gave any history of prior infection. Mention may also be relevantly made to the isolation of S. cholerae-suis from the faeces of a man whose infection proved rapidly fatal, and also from the faeces of his wife and child, who remained symptomless. . . . In other words, not only are there healthy carriers to contend with, but some types of Salmonella infection may give rise to clinical syndromes so mild as to pass unnoticed. Moreover, the convalescent carrier state may persist longer after food-borne Salmonella infection than has been generally believed.

Burt (1944), however, followed up one case for 4 years and *Salm. typhi-murium* was intermittently found over this period.

X-ray showed calculus and loss of gall bladder function. Finally, at operation, *Salm. typhi-murium* was isolated from the gall bladder membrane, the bile and centre of the gall stone. Following operation, all specimens were negative. See also Bornstein, Saphra, and Strauss (1941), and Borman, Wheeler, West, and Mickle (1943).

Rubenstein, Feemster, and Smith (1944) showed that no fewer than 6 out of 17 food-poisoning outbreaks were due to infection by human contact.

Glass, Goodheart, and Straker (1946) record that in an outbreak of food poisoning (a vanilla cream) caused by infected gelatin, the organism persisted in the stools of the patients as follows: 17 patients out of 32 excreted the organism for over 3 weeks; 16 over 4 weeks, 11 over 5 weeks, 7 over 6 weeks, 5 over 7 weeks, 2 over 8 weeks, and 1 for over 135 days. The patient excreting the organism for the longest period was a boy 9 years old. Tomlinson and Linsell (1946) found convalescents carried *Salm. thompson* for up to 6 weeks.

Grant (1950) noted the persistence of salmonella organisms in the faeces of 50 patients and 16 associated carriers: *Salm. typhi-murium*, 15; *Salm. thompson*, 13; and *Salm. stanley*, 3; the younger patients all being infected by *Salm. typhi-murium*, with the exception of 2 infected by *Salm. montevideo* and 1 by *Salm. dublin*. The carriers, both adult and children, were infected by *Salm. typhi-murium* except for one who harboured *Salm. thompson*. Of this group of patients and carriers more than half had ceased to excrete the organism within one month, but a fairly large proportion (11 per cent) carried that organism for over 3 months. See also Swan (1949), Cockburn (1950), Taylor (1950), Grant (1951).

Mackenzie (1951) records that during the previous 12 years the stools of over 1,600 men of the Metropolitan Water Board were examined. Of this total 450 were healthy individuals who had given suspicious agglutination reaction, and 1,150 had suffered from a recent attack of gastro-enteritis. No pathogenic organisms were recovered from the 450 healthy men. Of the 1,150, salmonellae were recovered from 10 individuals; 8 cleared up within 1 to 5 weeks, but 2 cases from which *Salm. typhi-murium* was isolated continued to excrete the organisms—one for 8 weeks and the other for 22 weeks. *Shigella sonnei* came from 9 individuals; of these 8 cleared up within 4 weeks but one continued to excrete this organism for 25 months.

Lennox, Harvey, and Thomson (1954) investigated a milk-spread outbreak due to *Salm. typhi-murium.* They found a slow rate of clearance of the organism from the faeces for 3 weeks, and then a rapid decline each week, leaving only 2 positives out of 60 cases at the end of 7 weeks, with persistence of one or two positives until 18 weeks. See also Miller, Nicol and Ramsden, (1955). It has been calculated that the salmonella carrier rates in different parts of the world range from 0·3 per cent to over 30 per cent.

Topley and Wilson (1955) remark:

> There is an increasing body of evidence to show that mild ambulant cases and latent infections resulting in faecal excretions of Salmonellae are quite common, and that chronic carriers, though proportionately fewer than those following infection with the typhoid bacillus do in fact occur.

Flies and Other Insects as Possible Carriers of Salmonella Infections

With regard to flies being possible vehicles of salmonella infection, it is well known that these insects assist in spreading many dangerous diseases, and that the organisms may be conveyed on their legs, wings, and bodies, or in their crops or intestines, but there appears to be no evidence that flies naturally harbour salmonella bacilli.

As showing how food and drink may be contaminated by house flies, Austen (1904), who carried out research on this subject, and whose writings concerning these insects are well known, in his article on 'The House-fly and Certain Allied Species as Disseminators of Enteric Fever', points out that during the South African and the Spanish-American Wars, thousands of cases of typhoid fever were traced to the contamination of food by flies.

Graham Smith (1914) found that with flies infected with *B. enteritidis*, this organism can be found in the contents of their crops and intestines at least 7 days after infection. A comprehensive study of bacteria on flies was recorded by Scott in America (1917). He found a seasonal variation in the bacterial content of flies in Washington, and that the greatest number of bacteria was found in the summer months (see also Nichol, 1917, Bishopp and Laake, 1921). Graham and his colleagues (1922) indicated that flies can transport type A toxin of *Cl. botulinum* on their feet and bodies, and also that it may be regurgitated from the crop material.

A number of interesting experiments were recently carried out in America by Ostrolenk and Welch (1942) on 'The House-Fly as a Vector of Food Poisoning Organisms in Food-Producing Establishments'. These were summed up by Savage (1942) as follows:

Specially reared flies were fed with infected food containing Salm. enteritidis, steps being taken to reduce surface contamination to a minimum. Not only were the flies readily infected but in transference experiments they transmitted the organism to fresh flies and these again to further series of flies. The organism was invariably isolated from the intestinal tracts and also from fly drinking water, food and the surfaces of the cages.

Longevity experiments demonstrated that this organism survived for at least 20 days and, if they had been carried further, probably for the entire duration of the life of the fly (4 weeks). Fly eggs planted in mash infected with Sal. enteritidis resulted in infected maggots, pupae and adults. Infected flies given access to healthy mice resulted in the infection of a number of the mice with some deaths. Further experiments also demonstrated that the transfer of the Salmonella from the infected mice to healthy flies was possible and took place in a number of instances.

Gwatkin and Mitchell (1944) conducted a number of experiments on the possible transmission of *Salm. pullorum* by house flies. The chicks used in these experiments were hatched from eggs obtained from a flock which had been free from pullorum disease for years. The flies were the common house fly (*Musca domestica*).

Gwatkin and Mitchell's summary is as follows:

In the first two experiments, chicks died from pullorum disease following access to feed contaminated by infected flies and to the flies themselves, some of which were probably eaten by the chicks.

In the third and fourth experiments, Sal. pullorum was not recovered from any of the chicks which had been given feed to which infected flies had had access. The chicks in Experiment 3 died as the result of having been chilled.

In the fifth experiment, however, the disease was produced in a small number of chicks by feeding chick mash which had been contaminated by infected flies. Some of the infected flies themselves were fed to another group of chicks and the organism was also recovered from a small proportion.

Subsequent failure to infect chicks by feeding or injecting relatively large amounts of culture suggests that the small number infected in the later experiments was probably due to lowered virulence of the infective agent.

Virulence of the infective agent would be better assured for this type of experiment of using organisms taken direct from chicks dead of the disease instead of cultures of the organism.

Sal. pullorum was recovered from the feet and wings of flies immediately after exposure and six hours later. It was recovered from the

gastro-intestinal tract up to five days, beyond which time examinations were not made.

Although the seasonal prevalence of house-flies and that of food poisoning is somewhat similar, the isolated nature of the outbreaks does not seem to favour fly-borne infection in this country. Under favourable conditions, however, it is possible that house-flies might convey dysentery bacilli responsible for this type of food poisoning.

Moorhead and Weiser (1946) endeavoured to prove the importance of the house fly (*Musca domestica*) for transmitting *Staphylococcus aureus* as a source of food poisoning. The flies, which had been without food for 24 hours, were fed with a growth of a food poisoning strain (611) of the organism on tryptone agar plates. The presence of *Staphylococcus aureus* (611) was identified in the digestive tract of such flies for 8 days after infection. The actual importance of this means of transmitting the organism in food poisoning could not be proved, but it is considered that flies may initiate and augment a food-poisoning outbreak. The possibility that *Staphylococcus aureus* may commonly be carried by flies was evidenced by the isolation of staphylococci from the digestive tract from 10 out of about 50 wild flies examined.

Alcivar and Campos (1946) carried out investigations in Guayaquil, South America, on flies as carriers of intestinal infections. The flies were caught in traps in the vicinity of the Institute. They were divided into lots of 20, and triturated in a mortar, emulsified in sterile saline (0·85 per cent). Tubes of media of different kinds were inoculated with the emulsion. Of 504 lots, 89 (17·5 per cent) were found positive, 46 (9·1 per cent) gave a growth of salmonella organisms (20 species), 60 (11·9 per cent) of shigella (6 species), and 17 (3·3 per cent) of both these. Salmonella organisms were isolated from 26 and shigella from 23 of 247 lots of *Musca domestica* (together 49 or 19·8 per cent), 11 salmonella and 15 shigella from 66 lots of *Chrysomyia macellaria* (together 26 or 38·4 per cent). From none was isolated *Salm. typhi* or *Shigella spigae*.

Watt and Lindsay (1948) conducted field experiments, lasting over two years, on the Mexican border of Texas, concerning the part played by flies in diarrhoeal diseases caused by shigellae or salmonella infections. The results showed that the control of flies by modern insecticides caused a significant reduction in the large amount of infection, illness and death due to diarrhoeal diseases in the area studied, especially those due to shigellae, but had little

effect on the incidence of salmonella infections. The trial showed, however, that control over flies had to be repeated frequently. The writers state that

> flies can be controlled by insecticidal methods, but the reversal of the fly curves in a matter of days after the treatment areas are changed shows all too clearly that chemical insecticides are temporary expedients at best. In our experience something more basic, particularly the elimination of man-made breeding places, must be done if the full effect of fly control on disease is to be brought about. The place that insecticides should occupy in a fly-control programme will not be clearly established until their use has been studied in conjunction with the various elements of sound municipal housekeeping.

Cockroaches

Mackerras and Pope (1948) reported that during an outbreak of salmonella infection at Brisbane, Australia, *Salm. bovis morbificans* and *Salm. typhi-murium* were isolated from cockroaches. The observers carried out experiments with four species of cockroach which were kept alive for long periods and fed with cultures of four different types of salmonella. All species of cockroach were susceptible to all salmonella types. A substantial proportion of the insects excreted the pathogen for more than a fortnight after a single infected feed. It is possible that cockroaches may play a part in the spread of salmonella infections and measures should be taken for their eradication when such infection is present in premises infested by these insects, which breed prolifically.

Bitter, Ruth, and Williams (1949) isolated various bacteria from the guts of 5 cockroaches (*Periplaneta americana*) collected in and around a hospital, a private house, and from sewer manholes in two towns in Texas. Among the organisms isolated were *Salm. paratyphi* B, *Salm. oranienburg*, and *Salm. bredeney*.

Olson and Rueger (1950) carried out transmission experiments to attempt an evaluation of the role of cockroaches as carriers of food infection organisms with *Salm. oranienburg* as the pathogen. Three species of common cockroaches were used. It was found that the test strain of salmonella could survive for 10 days in the faeces of the American cockroach and for 20 days in the Oriental cockroach. A post-mortem examination of the latter showed that it was positive 42 days after an infective feeding even though it had passed contaminated faeces only during the first 20 days. See also Macfie (1922), Wedberg and Clarke (1947), Mackerras and Mackerras (1948), Graffar and Mertens (1950), Janssen and Wedberg (1952).

Lice

Huang, Chang, and Lien (1937) reported that body lice removed from patients in China were found to be infected with *Salm. enteritidis.*

Ticks

Parker and Steinhaus (1943) infected ticks (*Dermacentor andersoni* Stiles) with *Salm. enteritidis* and found that these arachnids were capable of transmitting the infection to guinea pigs.

Fleas

Eskey, Prince, and Fuller (1949) carried out experimental investigations on the transmission of *Salm. enteritidis* by the rat fleas *Xenopsylla cheopis* and *Nosopsyllus faciatus*. The investigators demonstrated that the fleas may be infected with *Salm. enteritidis* when fed on infected mice and that they can transmit the infection from one mouse to another by their bites. The faeces of infected fleas also contain viable organisms in large numbers and provide an additional means by which the infection may be disseminated. Some fleas remain infected for more than 2 months. The mechanism by which the flea infects its host by its bite is unknown, but probably results from the regurgitation of infectious material from the eseophagus. See also Varela and Olarte (1946).

Organic Fertilizers and Salmonella Infections

Walker (1957) examined 123 samples of organic fertilizers purchased from retail shops and 50 (40 per cent) were found positive for salmonellae: 34 different types were isolated, all of which have been associated with infection in man, animals or poultry in this country; 28 types came from bone-meal, which was the most heavily contaminated fertilizer.

Walker in his summary remarks:

> Organic fertilisers may be a source of some of the unexplained salmonella outbreaks in man and animals. Moreover, since much of the raw material and some of the finished products (e.g. bone-meal) are imported from abroad, their use may also be responsible for the introduction in this country and the spread among animals of other diseases.

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Chapter VII

PREVENTION AND CONTROL

OWING to the world-wide distribution of the members of the salmonella group, a considerable number of which have been found pathogenic for man and animals, the prevention and control of salmonella infections now constitute a major problem for Medical Officers of Health, Medical Practitioners, Public Health Officials, Epidemiologists, and Bacteriologists.

It has been stated 'that to control infectious disease knowledge of its occurrence is the first line of defence'. In discussing hygienic and other preventive measures, several important relevant matters require special consideration.

Up to the end of 1939 food poisoning was not a notifiable disease, except in the County of London (Public Health (London) Act, 1936). In consequence, no reliable figures outside this area were available, and although from time to time the Ministry of Health issued valuable informative memoranda and advice dealing with the subject, many outbreaks and individual cases of food infection and intoxication occurred in the towns and rural districts which were never brought to light and consequently never investigated; moreover, unless each reported outbreak is systematically and scientifically studied, the information and data obtained often prove to be of little value.

This unsatisfactory state of affairs was remedied by the introduction of compulsory notification of cases of food poisoning, under the Food and Drugs Act, 1938.

In 1955, a new Food and Drugs Act was promulgated and in January 1956, the Food Hygiene Regulations came into operation.

NOTIFICATION OF CASES OF FOOD POISONING

Under Section 26, Part I, of the Food and Drugs Act (1955) we find:

(1) If a registered medical practitioner becomes aware, or suspects that a patient whom he is attending within the district of any local authority is suffering from food poisoning, he shall forthwith send to the medical officer of health of that district a certificate stating—

(*a*) the name, age and sex of the patient, and the address of the premises where the patient is, and

(*b*) particulars of the food poisoning from which he is, or is suspected to be, suffering,

and also stating whether the case occurs in the private practice of the practitioner, or in his practice as medical officer of a public body or institution.

(2) Where the local authority is not the local health authority the district medical officer of health shall send a copy of the certificate within twelve hours after its receipt to the local health authority.

Under Section 27, 'Inspection and Control of Infected Food', the Act states :

(1) If the medical officer of health of a district has reasonable ground for suspecting that any food of which he, or any other officer of the local authority of the district, has procured a sample under the provisions of this Act is likely to cause food poisoning, he may give notice to the person in charge of the food that, until his investigations are completed, the food, or any specified portion thereof, is not to be removed, or is not to be removed except to some place specified in the notice.

A person who uses or removes any food in contravention of the requirements of a notice given under this subsection shall be liable to a fine not exceeding ten pounds.

(2) If, as a result of his investigations, the medical officer is satisfied that the food in question, or any portion thereof, is likely to cause food poisoning, he may deal with it as food falling within subsection (1) of section nine of this Act and subsections (2) and (3) of that section shall apply accordingly; but, if he is satisfied that it may safely be used for human consumption, he shall forthwith withdraw his notice.

This compulsory notification should result in all cases of food poisoning being studied on the spot at the earliest possible stage, and comprehensive investigations made into the suspected sources and modes of infection, and enable the detailed results of the investigations and the confirmatory evidence of the bacteriological and pathological findings to be recorded, analysed, and classified. In this way much light will be thrown on unsolved problems of food-poisoning outbreaks and a vast amount of valuable and definite information gained. As time goes on, it should be possible to ascertain from this accumulated knowledge exactly how food becomes infected by members of the salmonella group and to discover with certainty the reservoirs, habitats, and paths of infection. This would allow of such exacting preventive measures being instituted as would eradicate food-poisoning outbreaks, or at least reduce their incidence to a minimum. In 1948, it was decided that Medical Officers of Health should include cases of food poisoning in their weekly and quarterly returns of notifiable infectious diseases. It has been suggested that coroners should be asked to report to the Medical Officer of Health of the district

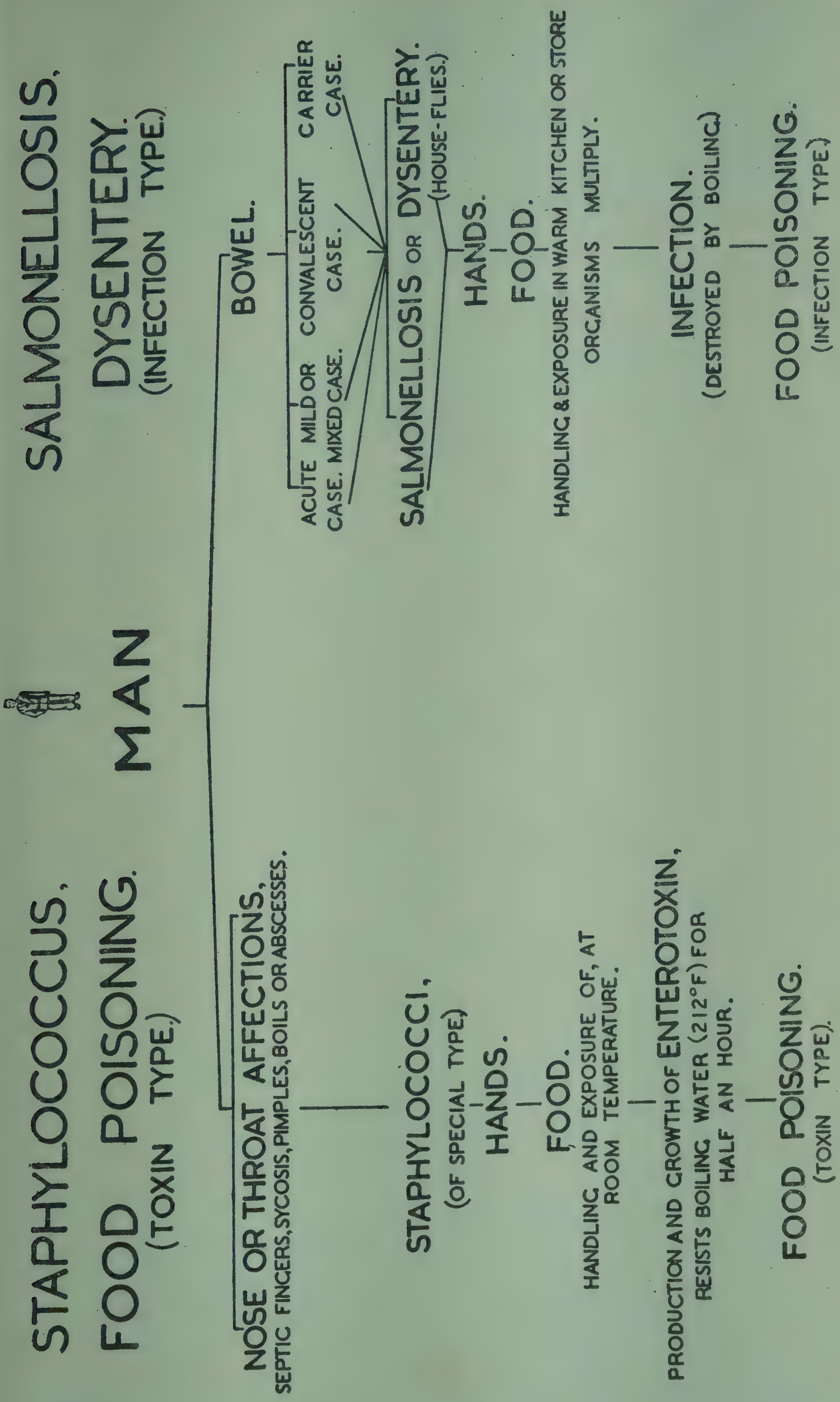

Fig. 2. Sources of Food Poisoning.

concerned, deaths of all persons upon whom inquests are held where the cause of death is associated with some form of food poisoning.

In the light of present knowledge, hygienic and other measures of prevention can only be adopted as will tend to control, to some extent, likely sources of infection. It must be remembered that the prevention of food-poisoning outbreaks is dependent upon the application of bacteriological knowledge of the organisms concerned to the practical problems of food distribution and food handling.

Legislation

The question arises, what are the existing legal measures for the general control of foods (including meat and meat foods) which should prevent or reduce contamination, unsound or infected foods reaching the consumer ?

The essential enactments which refer to food, i.e. its sale, adulteration, unsoundness (contaminated or infected), manufacture, handling, wrapping, transport, and storage, etc., are contained in the Food and Drugs Act, 1955, especially Sections Nos. 8, 9, 10, 11, 12, 13 (f), 22, and 23 of Part I, and 31 and 32 of Part II, and the Food Hygiene Regulations, 1955.

The Public Health (Meat) Regulations, 1924–52, together with the instructions on a 'System of Meat Inspection' issued by the Ministry of Agriculture, Fisheries and Food, Memorandum 3/Meat 1952 (based on the recommendations made in the Interdepartmental Committee's Report, 1953), constitute a valuable safeguard against the conversion of diseased or infected animals into human food. The provisions in the above Memorandum should help also to reduce the risk to public health by salmonella infections.

Among the most useful provisions of the Public Health (Meat) Regulations are Sections 8 (2), 9, 10, 12, 19, 20, and 21. Section 8 (2) requires notice to be given of the times of slaughter of animals, so that necessary inspection of the carcasses and offal can be made; also when sick or injured animals are to be or have been slaughtered for emergency reasons, the carcasses of which are for human consumption. In this connection it would be preferable, however, if the slaughter of sick or injured animals was excluded from private slaughter-houses because of the grave risk of transference of infection from sick animals to healthy meat.

In Section 10 of the Act,

> The person by or on whose behalf an animal is slaughtered for sale for human consumption shall not cause or permit the carcass of the

animal, including the mesentery and internal organs other than the stomach, intestines and bladder, to be removed from the place of slaughter until such carcass with its organs has been inspected, or its removal has been authorised by an Inspector of the Local Authority.

The Food and Drugs (Whalemeat) Regulations 1949–50, Memo. No. 1 deals with the inspection of whalemeat and the conditions under which whalemeat is prepared, etc., on floating factories, auxiliary vessels, and at land stations.

Topley and Wilson (1936) remark:

The hygienic precautions necessary to prevent food poisoning concern the whole course of the food from the slaughter of the animal to the final preparation for consumption. A thorough system of meat inspection is essential. The meat of animals that are ill or are emergency-slaughtered should, as a rule, be condemned. To this precaution alone Meyer (1916) attributes the comparative infrequency of meat poisoning in California where it is known that calves are infected with Salm. enteritidis.

Callow and Morgan (1938) pointed out:

As soon as an animal is killed, questions of hygiene, as they are commonly understood, become of paramount importance. Micro-organisms must be prevented from contaminating the carcass as far as possible. So far, however, no one has succeeded in devising an aseptic method of slaughter. Micro-organisms do obtain access to our meat, and all we can do is to devise means of decreasing their numbers and arresting their growth.

Contamination of meat is greatest on the slaughter floor, and it is at this point in particular that rigorous hygienic precautions are essential in order to produce meat with the maximum keeping qualities. The primary sources of infection are chiefly the feet, hides or skin, and the intestines of the animal. The infection is transmitted to the carcass by the hands, knives, swabs, washing water and clothing of the operatives, and, in fact it is reasonably well established that by comparison air-borne infection is negligible.

Control of temperature is the most important weapon that we have to reduce or prevent the growth of micro-organisms on meat. There are, however, other agents, including salting, smoking and drying, gaseous inhibitors such as carbon dioxide or ozone and ultra-violet light.

Transport and Handling of Meat and Offal

The Public Health (Meat) Regulations 1924 (d), Part IV, Sections 19, 20, and 21, on transport and handling have now been revoked and replaced by Sections 29 and 30, Part VI of the Food Hygiene Regulations, 1955, which deal with the transport of meat and offal and with the wearing of overalls by persons carrying meat, etc.

Regarding transport, the following requirements are recommended in the construction of a modern hygienic vehicle for the transport of meat carcasses, etc.

The vehicle to have a steel load-carrying frame welded to a low-slung chassis. The latter allows of easy loading and unloading and eliminates the need for steps or a ramp. The load is carried by the chassis and not by the body and so minimizes any swaying. The walls and roof should have inner and outer skins with sufficient space between them to allow for insulation. The interior of container to be lined with non-corrosive metal with corners rounded or curved, so as to ensure that no dust and dirt can collect and to facilitate cleansing. The metal floor should be of the corrugated type with channelling running longitudinally. Proper fitting sliding doors to be provided at each side of container and these to be kept closed, except during loading and unloading and cleansing operations. As there is no wood in this type of construction, the interior can be hosed down when required.

Permanent fly-proof and vermin-proof ventilation must be fixed in suitable positions.

Regarding suspension of the carcasses and to ensure even distribution of weight, suitably designed hanging gear, such as stainless steel rails and hooks, or steel rollers on an overhead endless track (part of the framework welded to chassis) should be installed. This system allows the carcasses to slide along the rail when loading or unloading with minimum effort to the meat porters and also reduces their need to traverse the floor of the vehicle.

It is preferable to use a separate vehicle for the transport of offal. Heads of animals may be hung up and offal placed in galvanized bins or other suitable non-corrosive receptacles (with close-fitting covers) which can be easily cleansed. Alternatively, sliding covered trays for offal could be fitted in the meat containers.

Knackers' Yards and Private Slaughter-Houses

The following acts and orders apply to knackers' yards. Section 12 (1) of Part IV of the Food and Drugs Act, 1955, says:

> No person shall sell, or offer or expose for sale, or have in his possession for the purpose of sale or of preparation for sale, for human consumption any part of, or product derived wholly or partly from, an animal which has been slaughtered in a knacker's yard or of which the carcase has been brought into a knacker's yard.

Section 13 (g) is concerned with

requiring the staining or sterilization in accordance with the regulations of meat which is unfit for human consumption, or which is derived from animals slaughtered in knacker's yards or from carcases brought into knacker's yards, or which, though not unfit for human consumption, is not intended therefor.

The Knacker's Yard Order, 1948, Supplies and Services Food (animals), requires the licensing of knacker's yards, the keeping of records of animal carcasses received, and the manner of their disposal. Martin (1950), referring to this important subject, remarks:

This is probably about as far as legislation can go, but considerable vigilance will always be necessary to prevent illicit trade. With animals killed at any odd time of the day or night, the meat taken into towns by fast motor vehicles and quickly disposed of, work of detection must inevitably be difficult. The provision of clearing-houses to which all meat coming into the towns from outside must be sent for inspection before exposure for sale, and a universal system of meat marking would of course solve the problem.

The supervision of privately-owned slaughter-houses is difficult even in well-organized centres, but the occupation of such buildings by unscrupulous dealers in a remote district is a menace to public health, more especially where the business of butcher and knacker is combined. There is no doubt that a certain amount of traffic in doubtful carcasses still goes on.

Public Slaughter-Houses

Sections 71 and 72 of the Food and Drugs Act, 1955, give authority to the local authority to provide public slaughter-houses and to make byelaws for securing that they are kept in a sanitary condition and properly managed. See also Circular by Minister of Agriculture, Fisheries, and Food on Slaughter-houses (Cmnd. 243, Aug. 1957) recommending minimum standards for the construction, lay-out, and equipment of slaughter-houses in England and Wales for the purpose of securing humane slaughter and hygienic conditions.

Before the decontrol of meat in 1954, the Government built several new and up-to-date slaughter-houses, which are operated by local authorities as public slaughter-houses. In addition two new public slaughter-houses have been built by local authorities. The minimum standards of structure, layout, and equipment, etc., of all slaughter-houses in England and Wales were proposed in a

report by the Inter-Departmental Committee on Slaughter-Houses, 1955. The standards should ensure humane treatment of animals, maintenance of hygienic conditions, and adequate facilities for meat inspection.

It is essential that slaughtering and inspection of food animals should be undertaken in the same place.

Leighton (1927) states:

> There may, or may not, be serious danger to the public from the meat of tubercular animals; emaciated carcases may, or may not offer risk to the consumer, but of all the cases which in our opinion throw upon the inspector a real responsibility and require great discretion in judgment, the most important are those which are killed to 'save their lives'.

Fourie (1936), referring to clinically sick animals, remarks:

> There should be no great difficulty in recognising many of the cases which could be responsible for food poisoning. The ante-mortem inspection for the recognition of such cases is of fundamental importance, as in many cases the naked eye appearance of the carcass and the lesions in the organs are such that they are not easily recognised. These are the dangerous cases, as the organisms may actually be present in the musculature, and under conditions of summer temperature may multiply very rapidly and produce their toxins in the meat.

Thornton (1957) points out:

> It is beyond doubt that many animals suffering from illness of an acute febrile nature are slaughtered in an attempt to market the carcase, and such cases add immeasurably to the responsibilities of the meat inspector as there are no facilities available for the bacteriological examination of the carcase and organs.

In some cases, however, this examination is carried out by the Public Health Laboratory Service.

The World Health Organization Monograph on Meat Hygiene, Series No. 33, recommends that any slaughter-house of importance should have its own laboratory where bacteriological and biochemical tests can be applied in suspected cases.

Bacteriological examination of suspected flesh, organs, and glands of food animals should, where possible, be carried out, although this seems hardly practicable as a general preventive measure. On the other hand, a bacteriological laboratory is an essential part of an up-to-date abattoir. In spite of all precautions at the time of slaughtering, meat infected with salmonella organisms occasionally passes the first line of defence and finds its way on to the market.

The ante-mortem examination, destruction, and burial of diseased animals is covered by the Diseases of Animals Act, 1950.

Other important orders are: the Tuberculosis (Area Eradication) Order, 1950; the Tuberculosis (Slaughter of Reactors) Order,1950; the Diseases of Animals (Waste Foods) Order, 1957.

Supervision of Meat Foods

The surfaces of flesh foods have a high water content and are subject to bacterial activity, especially during exposure in slaughter-houses, shops, markets, stores, transportation, etc., because of inadequate protection or refrigeration. Thus they furnish suitable media not only for the growth of non-pathogenic organisms, but for certain members of the salmonella group of bacilli. Research has shown that actual penetration of the bacilli below the surface, i.e. between the muscle fibres, deep into the meat, ordinarily takes some time to accomplish but is influenced by three factors—the temperature at which the meat is maintained, the amount of handling, and the time interval.

Savage and Bruce White (1925) in their study of 100 recent outbreaks of food poisoning state:

There are at least four reasons which justify and emphasise the need for special control. These are:

(*a*) They are foods made from pieces of meat, and therefore the chances of tracing the animal from which derived are limited. The great help afforded by an examination of the viscera of the animals supplying the meat is wanting.

(*b*) They are foods which are subjected to considerable manipulation and therefore are especially liable to bacterial contamination.

(*c*) They are mostly foods which are heated and then subjected to slow cooling, a procedure which facilitates and promotes bacterial growth in what is a suitable nutrient medium.

(*d*) They are varieties of foods the preparation of which is often carried out as an adjunct to other businesses, such as slaughter-house work either on the same or adjacent premises, which facilitate specific infection.

The 'made-up' foods here indicated include such foods as brawn, potted meat, meat pies, sausages. It is somewhat difficult to frame an inclusive definition.

The records of food-poisoning outbreaks show how important is this class of vehicle as a source of outbreaks, and how frequently their manufacture is associated with conditions which facilitate bacterial infection.

Regarding the importation of meat and offal from other countries, Hobbs (1957) points out:

There are many imported frozen meats and offal reaching us from other lands, a curious collection of substances to which far greater bacteriological attention ought to be given; not so much to whole

carcases but to the boned material and to offal. We may not suffer poisoning from meats sold by the joint or as chops for thorough cooking, but from the raw mince and raw sausages sold for the public to cook in a variety of ways and not always efficiently. A recent survey indicated that organisms could be isolated from a proportion of sausages from retail shops. If we knew the origin of the organism we would be a long way towards eliminating the contamination.

Recommendations for the Manufacture of Meat Products

The suitability and the hygienic conditions of premises used for the manufacture of meat products are of paramount importance. Preparation rooms, stores, and passages should be adequately ventilated and lighted, and proofed to prevent access of vermin and insects. No animals, or birds, should be allowed on the premises. Floors of rooms and passages should be constructed of impervious, non-slipping material which can be easily drained and cleaned; walls to be lined with glazed tiles to a height of five feet, but preferably to ceiling level; junction of walls with floors and ceilings to be coved. Ceilings to have smooth surfaces and constructed of material which will minimize condensation and be non-flaking to prevent dust and dirt (condensation can be obviated by means of hoods, screens, and ventilators); a constant supply of hot and cold water must be provided for cleansing utensils and equipment. A refrigerating plant or cooling chamber to be available for use in connection with the manufacture of open-packed meat products, which should be code-marked.

Equipment and utensils used in preparation and storage must be maintained in a cleanly condition and after use cleansed in boiling water containing a suitable detergent, or submitted to live steam. Separate sinks or tanks to be available for cleansing the above articles, apart from those sinks used in the preparation of meat products. Tables and similar fittings to have impervious surfaces.

Adequate cloakroom accommodation must be provided for the use of the staff and maintained in a cleanly condition. Lavatory basins should be situated as near the workrooms as possible and provided with supplies of hot and cold water, soap, and towels.

No smoking or snuff-taking should be allowed in the food preparation rooms. It is essential that handling of meat and meat products be reduced to a minimum to avoid possible contamination. The storage, handling, preparation, and processing of meat and offal *not* intended for human consumption should take place in rooms away from those in which the manufacture of meat products for human consumption is carried out. Animal casings

must be thoroughly cleansed in clean cold running water before being used.

As gelatine is one of the substances used in the manufacture of meat products, strict cleanliness must be observed in its handling, etc., because it is an excellent medium for the growth of organisms. It is preferable to use a gelatine with a low pH value (acid) in the manufacture of meat products.

The gelatine solution should be freshly made in sterilized utensils and used on the same day as it is prepared. The temperature of the solution not to exceed 140°F. as at higher temperatures changes occur in the gelatine. It loses its strength and setting properties and tends to decompose rapidly. It is very necessary that all food products containing gelatine should be cooled as quickly as possible after preparation to a temperature between 45° and 50°F. Pre-cooked meat for pies, if not used immediately, should be cooled to below 50°F. and kept at this temperature until required.

Personnel engaged in the manufacture of meat foods should be instructed in personal hygiene and wear washable overalls and caps during employment.

In 1949, a working party was appointed by the Minister of Food to review present trade practice and legal requirements for securing that conditions in the meat manufacturing trades are clean and sanitary and that the products and the materials from which they are prepared are wholesome in all respects. Their report was published in 1950.

Miller, Nicol, and Ramsden (1955) recorded a severe outbreak of food poisoning due to *Salm. bovis morbificans* (Basenau) which occurred in Lancashire after the consumption of meat pies. Over 1,000 persons were affected and 5 died. As a result of experimental researches carried out by Miller and Ramsden, certain recommendations and suggestions were made in their report in respect to baking practice as safeguards in the manufacture of meat pies.

The difficulties in tracing the original source of infection in food-poisoning outbreaks attributable to meat are obvious. During the various processes of handling and distribution, opportunities for contamination are many. Further difficulties arise in assigning a particular piece of meat to a specific carcass after it has been passed through a large retail or manufacturing establishment. Accurate records, therefore, should be kept, showing the original sources of the meat, viscera, etc., used in the manufacture of made-up food articles.

Legislation

Section 16 (1) of the Food and Drugs Act, 1955, deals with the registration by the local authority of premises used for the preparation or manufacture of sausages or potted, pressed, pickled, or preserved food for sale for human consumption.

Human Carriers

Anent this, the Chief Medical Officer of the Ministry of Health (1937) remarks:

The protection of cooked foods from infection at the hands of the vendors in shops has been much discussed, and especially in America bacteriological control to exclude 'carriers' from this occupation has been suggested. It seems plain, however, that any plan of this kind is impracticable, and that reliance should be placed rather on the provision of ample facilities for washing and on the inculcation of proper habits of personal hygiene among such persons. The increase in the number of shops selling cooked foods of all kinds, in urban districts especially, imposes on Medical Officers of Health the duty of careful supervision of their sanitary condition; they are probably the chief source of the increased prevalence of the intestinal infections.

Stebbings (1940), New York State Department of Health, has dealt with the general question of the examination of food handlers. He pointed out that it must involve a clinical history, physical examination, laboratory test and sometimes X-ray or other examination. He remarks:

This department is firmly convinced that, under the conditions in which it is possible to carry them out on a community basis, routine food handler examinations are unwarranted and are not to be considered a proper use for public funds.

Regarding the reduction of the risk of food infection from human carriers of salmonella, Savage (1956) remarks:

Chronic human carriers are rare, but with the present large numbers of outbreaks annually there must be at any one time a considerable number of people in the community who are for limited periods temporary Salmonella carriers. There is little risk of direct infection to other persons, but a real risk that they may infect foods which foster rapid bacterial multiplication and so become vehicles to spread food-poisoning. These risks occur in places where food is manipulated and where food is prepared for consumption, such as in catering kitchens.

Hobbs (1957) remarks:

In recent years a new method has developed for tracing carriers of organisms of the Salmonella group, that of the sewer swab technique. This method has been used with success to follow the course of a

polluted stream back to the source of pollution, which may be sewage from patients in a hospital or from an occupant in a private house (Moore 1948). It has also been used for the examination of waste in the drains and gullies of food manufacturing establishments when it is suspected that the presence of carriers or of contaminated foodstuffs may be a cause of trouble. (Phillips, 1956; Pyatt, 1956).

Administrative Measures

The Public Health (Infectious Disease Regulations) 1953, Part III, 4, 1–5, controls persons suffering from typhoid fever, paratyphoid fever, or other salmonella infections, dysentery, and staphylococcal infection likely to cause food poisoning. Food Hygiene Regulations, 1955, Part III, 11, 1–2, controls persons suffering from certain infections.

Preservatives in Food

The Public Health (Preservatives, etc., in Food) Regulations, 1925–53, make it an offence to manufacture for sale or sell any article of food which contains added preservative or colouring matter except as set out in the schedules of the above regulations. The preservatives allowed in certain specified foods, etc., are sulphur dioxide (sulphites) and benzoic acid (benzoates). With regard to colouring matters, simple and harmless agents such as cochineal and saffron may be used, but metallic colouring matters and coal-tar colours are prohibited. In the 1940 regulations (S.R. & O. No. 6, 33) the addition of sodium or potassium nitrate is permitted to bacon, ham, or cooked pickled meat, provided the total amount of nitrates in cooked pickled meat, other than bacon and ham, does not exceed 200 parts per million calculated as sodium nitrate.

A Sub-Committee of the Foods Standard Committee was set up in 1951 to review the existing Public Health (Preservatives, etc., in Food) Regulations and to make any recommendations considered desirable for their amendment. A report was issued in 1954 by the Foods Standards Committee recommending that the regulations be amended to authorize the addition of anti-oxidants to certain foods, such as edible oils and fats, but not including butter, as follows:

Antioxidant	*Edible oils and fats per cent*	*Essential Oils per cent*
Propyl, or octyl, or dodecyl gallate, or any mixture thereof	0·01	0·1
Butylated hydroxyanisole	0·02	0·1

Butylated hydroxyanisole (up to 0·2 per cent) may be used in conjunction with the gallates (up to 0·01 per cent) in edible oils and fats; but in the case of essential oils the total amount of antioxidant or mixture of antioxidants should not exceed 0·1 per cent.

In 1955, the Foods Standard Committee issued a supplementary report on the recommendations relating to the use of colouring matters in foods, and also a report in 1956 on emulsifying and stabilizing agents in foods. The Colouring Matter in Food Regulations (S.I. 1957, No. 1066) were issued in June 1957.

Cooking of Foods

Although the various processes for cooking may fail to destroy all organisms, they doubtless diminish the number considerably, but much depends on the consistency and structure of the foods. Those organisms surviving in sound, freshly, and thoroughly cooked food are not ordinarily a menace. The consumption of such food is less liable to cause poisoning than raw or partly cooked food. Non-acid foods which are freely handled during preparation and served without being cooked, or only partially cooked, and afterwards allowed to cool at room temperature, are often the responsible vehicles in food-poisoning outbreaks. Whatever danger there may be, either from infection or intoxication, is dependent upon storage with the incubation or re-infection of the cooked product. Food which is to be re-heated should be brought to, or higher than, boiling point to kill off contaminents.

Meyer (1953) remarks:

> Food to be eaten raw must be fresh and free from stale odours and from soft or decomposed areas and must be thoroughly cleaned and washed in drinking water; secondly, sound food must be thoroughly cooked in small pieces and long enough to heat the centre of the pieces, use of thermometers in restaurants, institutions and kitchens being of real value. Soft cooked foods, if not eaten promptly, must be refrigerated or recooked before serving, even if signs of spoilage are not evident ; and reconstituted egg powders should be cooked immediately and thoroughly after reconstitution.

Refrigeration

For many years action of cold has been extensively used for the preservation of perishable foods and drink of every description, during transport by road, ship, rail, and while in storage. Today refrigeration is one of the most widely used methods, not

only for preserving meat and other foods commercially, but also in the home. The method which extracts heat from the foodstuffs by mechanical or chemical devices probably has less prejudice to contend with than any other process for temporarily preserving foods. It enables the comestibles to be maintained in a sound and palatable condition, the nutritive properties are not substantially altered, and only a slight loss of moisture occurs; moreover it provides a uniform temperature of 30°–50°F., low enough to prevent the growth of organisms.

The minimum temperatures at which the most important contaminating food organisms will grow are approximately: micrococci 42°F., *Proteus vulgaris* and *Staphylococcus aureus* 50°F. or lower, coli-aerogenes group, salmonella, 60°F. or lower. Optimal temperatures are, of course, considerably higher.

Refrigerators, however, have certain limitations. They are not sterilizers and cannot render food safe if it has been infected by pathogenic organisms. These may multiply upon the meat or other food during the time it is out of the apparatus and be rendered temporarily inactive but not destroyed by refrigeration. They will rapidly multiply if the food again reaches a suitable temperature. The film of moisture caused by the 'thawing' of 'frosted' meat provides a suitable medium for bacterial activity, and increases their penetrating power. The moisture content is often more real than is probably noticeable. Dry conditions are unfavourable for the multiplication of organisms, though not necessarily for their survival. It is important, therefore, that the walls and air inside a refrigerator be as dry as possible. Moulds which cause spoilage develop very slowly at a temperature of 50°F., but grow rapidly at higher temperatures, especially on moist surfaces. It is essential that the refrigerator should be kept clean.

Before starting up a new apparatus there are certain precautionary measures which require attention. The inside walls and floor should be washed with warm soap-and-water and the shelves, ice-trays, and cooling radiator with warm water only. In warm weather, the room where the apparatus is installed should be well ventilated, especially during the night. The food must be clean, as fresh as possible, and not handled more than is necessary. It is important that 'left-over' foods, such as meat stews, brawn, creamed vegetables, etc., be cooled to a temperature below 40°F. Ordinarily food is not wrapped, but placed in the apparatus so that the cold air circulates freely round it. No hot food should be placed in a refrigerator to cool.

Recommended temperature ranges: maximum acceptable temperature for storage of all perishable foods, 50°F.; dairy products, 38°–46°F.; meat and fowl, 33°–38°F.; fruits (except bananas) and vegetables, 44°–50°F.; fish and shell-fish, 23°–30°F.; frozen goods, 0°–20°F.

Faults in the operation of refrigeration may consist of too frequent opening of the doors, giving rise to temperature fluctuations; overcrowding and poor dispersal of foods in the refrigerator, causing interference with air circulation. When a refrigerator is packed closely with warm meat, the temperature in it may not fall to a safe level for many hours. Instances have occurred where the circulation of the air was blocked and the meat near the cooler frozen, while in the rest of the box the air was stagnant and the meat warm and moist. Improper or faulty connections of drains and lack of attention to odours and cleanliness also cause trouble. When defrosting, or when the refrigerator is empty, the inside should be thoroughly cleaned with warm soap and water, more especially if spoiled meat has been found in the refrigerator.

Grundy (1952) records an occurrence at Wembley which indicates how the interruption of electricity supplies to refrigerators may be responsible for outbreaks of food poisoning.

> Meat was obtained from a factory canteen, cooked on Friday the 19th October and braised, then placed in the refrigerator for use on the following Monday. Unfortunately during the weekend there was an electrical breakdown, which necessitated a switch-off for all current, allowing the meat to remain at a much higher temperature than was intended. The consumption of this meat in a made-up dish on the Monday resulted in 60 cases of food poisoning, every single person who consumed any of the affected food. The cases fortunately were mild, with diarrhoea and abdominal pain, but the incident illustrates the importance of using prepared meat immediately after cooking and never delaying its use to some future time.

Milk-Borne Infections

The prevention of these infections may be effectively accomplished by efficient pasteurization or other adequate form of heat treatment of the milk. After pasteurization the cooling process is most important, in accordance with the Milk (Special Designation) (Pasteurized and Sterilized Milk) Regulations, 1949.

Cleanliness alone is not a safeguard against infection conveyed by the milk of a diseased cow, and such milk may even pass the routine bacteriological standards for cleanliness. This is referred

to by the Chief Medical Officer of the Ministry of Health in his report for 1934 as follows:

Cleanliness, however, is important from an aesthetic and commercial standpoint. Dirty milk is not only aesthetically objectionable but it has also poor keeping qualities, and for this reason alone reputable firms are anxious to obtain their supplies as clean as possible. Whilst, therefore, cleanliness is desirable, cleanliness is not enough. Safety is the really important consideration, and in present circumstances the ordinary raw milk supply can never be regarded as safe. To ensure its safety, that is to say, its freedom from pathogenic organisms, suitable heat treatment such as that afforded by efficient pasteurisation is essential.

Dolman (1941), writing on the status of milk-borne hazards, believes that the actual incidence of disease is higher than that recorded, but no very close estimate of the former in terms of the latter can be made, owing to the many weak or missing links in the chain of evidence. He is of opinion that the incidence of milk-borne disease may be decided for all practical purposes by the following three main groups of variables:

1. The distribution among dairy cattle of tuberculosis, Bang's disease, and of streptococcal and staphylococcal mastitis.
2. The extent to which handlers of milk and milk products may be infected with, or may carry, micro-organisms of the typhoid-paratyphoid-dysentery group, Group A streptococci, enterotoxigenic staphylococci, and diphtheria bacilli.
3. The extent to which supplies of milk and milk products are effectively pasteurized.

Marked fluctuations in any component of these groups of variables will inevitably be reflected in the apparent trend of the incidence of milk-borne disease.

Dolman considers staphylococcal mastitis is of much greater importance from the point of view of public health than streptococcal mastitis.

Watts (1947), writing on the importance of cowshed hygiene in transmission of milk-borne disease, draws attention to the following:

The form of mastitis of greatest public health interest is that caused by human pathogens. These are usually streptococci (Wilson 1933 and 1942) and give rise to epidemics of septic sore throat or scarlet fever. Their origin is the attendants and therefore it is broadly true to say that, with the possible exception of gastro-enteritis due to the dublin type of *Salm. enteritidis*, all milk-borne diseases amenable to hygienic precautions are of human origin. The rôle of hygiene in the cowshed is therefore the prevention of the infection of the milk or cow by human pathogens from the attendants.

The usual cause of gastro-enteritis, due to direct cow infection, is *Sal. enteritidis var. dublin*. It was supposed that this was present in the udder, but recently Rankin and Slavin (1947) have suggested that milk is infected by faecal contamination and that the seat of infection is the gall bladder. If this is so, modern hygienic measures should reduce the incidence of this disease, as in fact it appears to have done. . . . Assuming that the usual clean milking practices are adopted, I think there are two common faults: (1) excessive handling, and (2) inefficient washing.

Regarding (1) Watts advocates the use of the milking machine, where the milk is automatically removed to the cooler and thence to the churns. In that case, there is no need to handle the cow during the process. Regarding (2) he is of opinion that this is due to one or more of the following factors: use of weak disinfectant; use of only one or two washing cloths; washing too many animals with one bucketful of disinfectant; washing too much of the animal.

While the first three need no explanation, the fourth perhaps does. In hand-milked herds it is essential that the udder and flanks of the animal should be clean. Otherwise particles of dirt would fall into the pails. This has led to the washing of these parts of the cow. In machine-milked herds with totally enclosed units there is no such risk. The greater risk is that the dirty cloths and exhausted disinfectant which have been used for such washing are then applied to the teats. In addition, the 'liquid' left on the udder runs down to the most pendulous points, the teats. I therefore suggest that only the teats should be washed before milking. If necessary, the udder and flanks can be washed *after* milking but never *before*. Not only will this prevent faecal contamination of the milk, causing such diseases as gastro-enteritis, but it will also reduce the rate of destruction of the disinfectant, since the amount of dirt on the teats is far less than that on the udder and flanks.

Smith (1955) remarks:

> The transmission of disease to man through the agency of milk and milk products appears to have become a less frequent and less serious menace. The lessening of the incidence of milk-borne disease is particularly noticeable with the infections which do not occur naturally in animals whose milk is used by mankind, though some of the natural infections of dairy cows, such as tuberculosis has also been greatly reduced. The marked improvement in dairy hygiene and dairy techniques has lessened the opportunities for man to infect dairy animals with certain organisms. Moreover, with the lessening incidence of infectious disease in man there has been a marked reduction in the number of certain types of carriers. This, together with the increasing use of pasteurization has reduced greatly the possibility of infecting milk.

Paper Containers for Milk

Albeit these have been available for a considerable time, only in recent years has the dairy industry begun to use them, and this

has instigated the development of methods to ensure their sanitary quality.

Incidentally paper containers for fresh milk have the advantage of conserving space and weight. Twelve quarts of milk in glass bottles weigh approximately 60 lb. The same milk in paper containers weighs only 25 lb. and occupies half the space required for glass containers.

In the United States, where their use is rapidly increasing, the New York State Agricultural Experiment Station has been studying the sanitary condition of paper stock used for milk containers. Subjoined are their recommendations regarding the production and handling of these to prevent infection of the milk:

(1) Use of virgin pulp only.

(2) Pure process water and strict microbiological control of pulp and paper mills.

(3) Suitable protection and wrapping of finished board.

(4) Mechanical handling of board and containers at conversion factories and milk plants.

(5) Protection of board, adhesives, moisture-proofing materials, and finished containers, from careless exposure to human contact, contamination, dirt, flushing water, or insects.

(6) Detailed knowledge and careful selection of all materials composing the container, to avoid the possibility of incorporating substances having germicidal or bacteriostatic effects, the use of which is prohibited unless they have been shown to be non-toxic to human beings and without effect on milk.

The Geneva Conference, 1938, suggested that

Board prior to moisture-proofing shall not, at any time, exceed 500 colonies per gram of disintegrated board, and the average bacterial content of finished containers should not exceed 50 colonies per container.

These standards are lenient, and workers in this field have shown that the average container on the market will meet them easily.

Davenport (1955–6) remarks:

The suitability of a glass bottle as a milk container is open to challenge. With modern manufacturing methods there seems no reason why this danger should be tolerated. Plastic containers or disposable waxed cartons are in constant use for other food-stuffs why not for milk? The waxed carton is already used and approved, even in this country. It has the advantage of cleanliness, safety, and it needs no collection, washing and sterilization after use. The question of cost need not be formidable, as a recent survey in Sweden showed that wax milk packaging could be operated for as little as 0·19d. per pint more than for glass bottles.

Animal Vectors in Milk Outbreaks

It has been suggested that in outbreaks of food poisoning connected with milk and milk products, a possible animal vector should not be overlooked, and that veterinary co-operation should be sought in order to secure a prompt investigation into the question of infection from a bovine source. The routine inspection of dairy cattle is carried out by veterinary inspectors appointed by the Minister of Agriculture, Fisheries and Food, under Section 12 of the Food and Drugs (Milk, Dairies and Artificial Cream) Act, 1950, and Section 34, Part II, of the Food and Drugs Act, 1955.

Legislation

In the Milk and Dairies Regulations, 1949, Part IV deals with the inspection and health of cattle; Part VI, special provisions applicable to the production of milk and the treatment, handling, and storage of milk; Part VII, provisions with regard to infection of milk; Part VIII, general provisions for protecting milk against contamination or infection; Part IX, provisions relating to the cleansing and storage of vessels, utensils, and appliances; Part X, conveyance and distribution of milk; Food and Drugs Act, 1955, Part II, Sec. 31, Prohibition of Sale of Milk from Diseased Cows.

The Public Health (Infectious Diseases) Regulations, 1953, Part III, empower the exclusion of a carrier of typhoid fever, paratyphoic fever, or other salmonella infection, or dysentery or staphylococcal infection likely to cause food poisoning, and details measures to prevent the spread of infection.

Milk Products—Cheese

A number of food poisoning outbreaks have been recorded from time to time, at home and abroad, due to cheese infected by salmonellae, or to contamination by staphylococcal organisms. Savage and Bruce White (1925) proved experimentally that salmonella strains survived in cheese up to 30 days. Minett (1938) showed that entertoxin subsisted in cheese made with raw milk. Erkman (1941) refers to 21 outbreaks of food-poisoning type in Turkey due to cheese. Meyer (1944) stated that 'there is only one way to render cheese safe, and that is by pasteurisation'. Berbereau (1946) records a number of outbreaks in Syria and Lebanon due to locally prepared green cheese contaminated in most cases by *Staphylococcus aureus*.

Tucker, Cameron, Henderson, and Beyer (1947) describe an outbreak which involved 250 cases from six towns in West

Tennessee, 100 cases in Lawrence County, Ill., and 34 cases in Fulton County, Ky., due to the consumption of colby cheese (similar to cheddar cheese). The illness was severe but no deaths occurred. The symptoms were sudden onset with chills, fever, nausea, vomiting, and diarrhoea, lasting 3 to 10 days. *Salm. typhi-murium* was isolated from the stool of one patient and from some of the cheese. In all the widely distributed cases there was the common factor, that all the patients had eaten cheese 24 to 48 hours before the onset of the illness. Members of those families who did not eat the cheese remained unaffected. The cheese was all derived from a creamery in Illinois and was made on 12 March, 1945. The cases began on 26 March, the cheese having been distributed and sold soon after manufacture. It was stated that 'the head cheese-maker found a mouse floating in the vat, fished it out and completed the filling of the vat'. The milk used was not pasteurized.

The authors obtained some of the cheese and investigated the viability of *Salm. typhi-murium* in it. The cheese was held in an electrical refrigerator unit at 43° to 48°F. and the organism remained viable for 302 days.

Fabian (1947) records that during the past 50 years or so there have been no less than 59 outbreaks of disease with 2,904 cases and 117 deaths in the United States and Canada which were traced to the consumption of cheese. The organism most commonly associated with cheese-borne infections are members of the salmonella group, such as *aertrycke*, *schottmulleri*, *typhosus*, *suipestifer*, *typhi-murium*, and *cholerae-suis*; staphylococci such as *Staphylococcus albus* and *aureus*; of the brucella group, *Brucella melitensis* but not *abortus*; and *Clostridium* botulinum.

Fabian states that

two things stand out when a study is made of the various epidemics caused by cheese. One is that the cheese generally has been made from raw milk and that it has been sold and eaten too quickly after it has been made. Likewise, in the experiments testing the longevity of pathogens in cheese, the data show that pathogenic bacteria die out more quickly at high than low temperatures. This leads to the obvious conclusion that all milk and cream used in the manufacture of cheese should be pasteurised and aged at a high temperature. If this were done, the transmission of disease by eating cheese would disappear.

Ice-Cream

Bacteriologists have stressed the danger of the infection of bulk ice-cream during dispensing. The factory-filled package has been

recommended as a means of avoiding possible contamination. Paper cups, boxes, bags, etc., used for ice-cream, must be properly protected during storage and handling. All containers in factories should be assembled with as little handling as possible. There should be frequent sterilization of plant and utensils in ice-cream factories.

'Ageing' or slow cooling should take place in a separate room to that used for mixing and freezing. All ingredients used must be adequately protected against contamination during storage.

The following sections of the Food and Drugs Act, 1955, refer to ice-cream: Part I, Section 22 (1), (2), and (3), 'Sale of ice-cream from stalls'; Part I, Section 23 (1) to (5), 'The prevention and spread of disease by ice-cream'.

The Ice-Cream (Heat Treatment, etc.) Regulations, 1947, came into operation on 1 May, 1947. Outbreaks of food poisoning due to the consumption of infected ice-cream had occurred from time to time and it was realized that the manufacture, handling, transportation, and sale of this commodity was an important branch of the food industry. Hence the necessity for the Regulations, the main provisions of which are subjoined:

In these regulations 'ice-cream' includes water ices and any article, under whatever description it is sold, which is so similar to ice-cream as to constitute a substitute therefor; 'ingredients' includes sugar and dried egg, but does not include colouring or flavouring materials or fruit, nuts, chocolate, and other similar substances; and 'complete cold mix' means a product which is capable of manufacture into ice-cream with the addition of water only, is sent out by the manufacturer in airtight containers, and has been made by evaporating a liquid mixture which has already been submitted to heat treatment comparable with that prescribed in these regulations

The following requirements shall be observed in the manufacture of ice-cream intended for sale for human consumption:

1. Where a complete cold mix is used which is reconstituted with wholesome drinking water and to which nothing is added other than colouring or flavouring materials, fruit nuts, chocolate or other similar substances, the reconstituted product shall be converted into ice-cream within 1 hour of reconstitution.
2. In any other case, after the ingredients have been mixed together the following provisions shall apply:
   (*a*) the mixture shall not be kept for more than one hour at any temperature which exceeds 45°F. before being sub-

jected to heat treatment in accordance with the next following sub-paragraph

(*b*) the mixture shall be subjected to heat treatment as follows: it shall be raised to and kept at a temperature of not less than 150°F. for 30 minutes or alternatively of not less than 160°F. for 10 minutes

(*c*) after the mixture has been subjected to heat treatment as aforesaid it shall be reduced to a temperature of not more than 45°F. within $1\frac{1}{2}$ hours and shall be kept at such a temperature until the freezing process is begun

(*d*) such indicating and recording thermometers shall be used as the local authority considers requisite for indicating and recording the temperatures to or at which the mixture is raised, kept or reduced

(*e*) the records of any thermometers used to record the temperatures to or at which the mixture is raised, kept or reduced shall be preserved for a period of not less than one month

(*f*) all apparatus used for the purposes of this paragraph shall be installed, maintained and operated to the satisfaction of the local authority.

Ice-cream shall not be sold or offered for sale unless either:

1. it has been kept at a temperature not exceeding 28°F. since it was frozen, or
2. if its temperature has risen above 28°F. at any time since it was frozen, it has again been subjected to the treatment prescribed by sub-paragraphs (*a*), (*b*) and (*c*) and, having again been frozen, has been kept at a temperature not exceeding 28°F.

Ice-cream shall be protected from dirt, dust, or other contamination at all times during its manufacture, storage, and distribution and all apparatus and utensils brought into contact with ice-cream during its manufacture, storage, or distribution shall be thoroughly cleansed immediately after use and shall be kept clean at all times.

Other statutory instruments are as follows:

The Food Standards (Ice-cream) Order, 1951 (also 1953). This prescribes the minimum standards of composition of ice-cream.

The Ice-cream (Heat Treatment, etc.) Regulations, 1947–51.

The Ice-cream (Heat Treatment, etc.) Amendment Regulations, 1952.

Since the Heat Treatment Regulations came into force, only

a few minor outbreaks of food poisoning incriminating ice-cream have been reported (Hobbs, 1951).

It has been suggested that the hygienic quality of ice-cream can be obtained by performing a total bacterial count, a coliform count, and the identification of the coliforms as of excremental type or otherwise. The tests of the heat-treated product may

OUTBREAKS OF FOOD POISONING AND FOOD-BORNE INFECTION IN ENGLAND AND WALES CAUSED BY ICE-CREAM

Date	*Place*	*Disease*	*Infective Bacterium*
1892	London	Typhoid fever	*Salm. typhi*
1904	Govan, Glasgow	Typhoid fever	*Salm. typhi*
1910	Eccles	Typhoid fever	*Salm. typhi*
1916	Boldon Colliery	Typhoid fever	*Salm. typhi*
1933	Glasgow	Dysentery	*Sh. sonnei*
1935	Lanarkshire	Dysentery	*Sh. sonnei*
1935	Grays-in-Thurrock	Typhoid fever	*Salm. typhi*
1937	Glasgow	Diphtheria	*C. diphtheriae*
1937	Southampton	Paratyphoid fever	*Salm. paratyphi* B
1945	Oxford	Food poisoning	*Staph. pyogenes*
1945	Oxford	Food poisoning	*Staph. pyogenes*
1945	Oxford	Food poisoning	*Staph. pyogenes*
1946	Aberystwyth	Typhoid fever	*Salm. typhi*
1946	Lincoln	Food poisoning	*Salm. typhi-murium*
1947	Winchester	Food poisoning	*Salm. typhi-murium*
[1947	*Ice-cream (Heat-Treatment, etc.) Regulations*]		
1949	Manchester	Food poisoning	*Str. faecalis*
1949	Bradford	Food poisoning	*Salm. newport*
1954	Cardiff (cold mix)	Food poisoning	*Staph. aureus*

indicate whether the 1947 regulations really afford an adequate safeguard. The neglect of elementary precautions in handling and in ensuring the cleanliness of utensils can still lead to serious contamination if only in less degree.

In a report of the sub-committee of staff committee of the Public Health Laboratory Service, by Gillespie, King, Macdonald, Moore, and Tomlinson (1947), is given an account of an investigation into the bacteriological examination and grading of ice-cream. The technique for the examination and collection of samples also is described. If the ice-cream is sold in wrapped blocks or in cartons, the sample should consist of one block or carton. When taking samples of other forms of ice-cream, the retailer's server or spatula should be used and the sample placed in a sterilized 2 oz. (fluid) wide-mouth glass stoppered bottle or jar or other suitable container and filled so as to leave as little free air space

as possible. The sample, which should be collected from the surface of the ice-cream, must be kept as cool as possible and reach the laboratory within 2 hours. Where this is not possible, the sample must be packed with ice (or solid carbon dioxide) in a container and forwarded to the laboratory within 6 hours from the time of taking the sample. If on arrival at the laboratory the ice-cream has melted, it should be placed in a refrigerator till the examination is carried out. If sample has not melted, then allow it to stand at room temperature for a maximum period of 1 hour till it is melted.

In the Second Report of the Sub-Committee on the Bacteriological Examination and Grading of Ice-cream, Gillespie, King, Macdonald, and Tomlinson (1948), under the heading, 'Unhygienic conditions of production', remark:

Much of the low grading of ice-cream is associated with faulty cleaning and sterilising of plant, especially the equipment used after the ice-cream has been heat-treated, namely, the pipes and valves going to the homogeniser, cooler, storage tanks, and the freezer. Special attention must be given to the cooler because of the large surface exposed and of the crevices where dirt can remain, and to the freezer because of the blades.

In heat-treated ice-cream a frequent cause of trouble is delayed cooling. Ice-cream must be cooled to 45°F. or below within 1½ hours, according to the regulations, to prevent bacterial multiplication and subsequent deterioration in quality. Owing to shortages in supplies, there are many firms that have no cooling equipment whatever.

In cold-mix ice-cream the common source of trouble is faulty sterilisation of utensils such as the mixing bowl, spoons, and the freezer with its blades. Some type of sterilisation must be used, as cleaning alone is not a sufficient guarantee against contamination and multiplication.

In pre-packed ice-cream the method of pre-packing must be carefully scrutinised, as many of the cutting and packing machines are difficult to clean and sterilise. Hand pre-packing has its own obvious dangers.

Retail samples are frequently contaminated by the serving spoons or spatulas, which are usually left on the counter or in a bowl of water. Though the water may be boiled originally once it is enriched with ice-cream it affords an excellent culture medium for bacterial multiplication, and in warm weather soon becomes grossly contaminated with micro-organisms.

In the summary and conclusions of the Fourth Report of the Sub-Committee (Gillespie, King, Moore, and Tomlinson, 1950) on the bacteriological grading of ice-cream, it is stated:

(1) The results of the bacteriological examination of ice-cream samples by the methylene blue reduction test during 1949 confirm our previous conclusion that the test affords a simple and practical means for the routine grading of the bacterial cleanliness of ice-cream.

(2) To obviate confusion in recording the result of the test it has been decided that any sample which fails to reduce methylene blue completely in four hours shall be classified as Grade I.

(3) It has previously been pointed out that the results of the presumptive coliform test cannot be accepted as evidence of excretal pollution, since only a small proportion of these organisms belong to the faecal type, Bact. coli. Type I.

(5) It may therefore be concluded that the assumed value of the coliform test in assessing the safety of ice-cream is without adequate scientific foundation.

Illustrative Outbreaks

Williams, Swift, Vollum, and Wilson (1946), as reported by Savage, describe in detail three outbreaks of staphylococcal food poisoning due to ice-cream. The essential facts regarding these outbreaks are as follows:

First outbreak: Oxford City.—The vehicle was ice-cream, but as over 500 gallons were made, and as there were only 40 cases, it was obvious that only a small portion was infected. Most of the ice-cream was home-made, but 120 gallons came from a U.S. Army hospital in various containers by lorry. The infected ice-cream appears to have been 2 gallons of this mixed with 10 gallons of the maker's own cold-mix ice-cream. Only persons known to have consumed ice-cream from this batch were made ill. The U.S.A. ice-cream was prepared from sugar, powdered milk, dried egg, cornstarch and vanilla, the sugar and cornstarch being first heated to boiling point. The hot-mix was allowed to cool at room temperature, and had remained in the warm kitchen overnight until despatched to Oxford about 8 a.m. next morning. Bacteriological investigations of patients yielded *staph. aureus* type 42 D. This type was found in the American ice-cream in large numbers, in the American and Oxford ice-cream mixture also in large numbers, but not in the Oxford vendor's own ice-cream. It was also isolated from the nose and hands of one of the assistants, Pte. G., who prepared the ice-cream mix in the U.S. hospital. This man was probably the source of infection, and the long period of slow cooling allowed abundant multiplication of staphylococci, and enterotoxin production. The same type 42 D was found on the hands of the Oxford proprietor and those of the two roundsmen, and in a dried egg packet, but as all had handled the infected ice-cream this was probably the source of infection. The incubation period was 2–3 hours, the symptoms were of the usual enterotoxin type and all the patients quickly recovered.

Second outbreak: U.S. Hospital.—This occurred 10 days after the first outbreak. The vehicle was ice-cream consumed in the hospital and served to about 1400 persons, patients and staff, of whom some 500 to 600 were attacked with food poisoning after an incubation period of $2\frac{1}{2}$–3 hours. Symptoms were similar to those in the previous outbreak, but rather more acute and severe. Most of the patients recovered by the next day, but weakness persisted longer in some cases. Only those who

consumed the ice-cream were attacked. The mix, of the same composition as in the first outbreak, was prepared on the evening of April 5th and was, as before, kept in the warm cookhouse from its completion at midnight until 5 a.m. next morning; it was then taken to the Oxford vendor to be frozen (done on evening of 6th) and was taken back by lorry on the morning of April 8th, having been kept in a refrigerator in the meantime.

Laboratory investigations yielded from patients the same *Staph. aureus* type 42 D, and this was also found in the remains of the ice-cream. All strains were coagulase-positive. Investigations at the Oxford premises showed that the infection did not originate there. The facts strongly suggested that the same Pte. G., responsible for the first attack, also infected this ice-cream, and the same reprehensible slow cooking at room temperature was again allowed. Pte. G. could not be examined for *Staph. aureus* as he had been transferred to another unit.

Third outbreak: Oxford City.—This outbreak in May 1945, also due to ice-cream, occurred in Oxford and in an institute at Botley. Exact number of cases are not known but 58 patients received treatment at the Radcliffe Infirmary. The incubation period was 2 to 4 hours, and the symptoms were as in the other two outbreaks. The ice-cream eaten over the period came from two sources, one was made locally by the same Oxford producer and dealer, and the other came from a U.S.A. Infantry camp in Wiltshire. Detailed study showed that only that produced on the camp premises was infected, and this was fully confirmed by the fact that the hospital pathologist and 16 other men, who consumed some of it from a can of the U.S.A. product returned to the camp, all suffered from food poisoning. The ice-cream had the same composition as in the other outbreaks and again was allowed to cool down in the warm cookhouse after heating. It was exposed to atmospheric temperature for approximately 18 to 32 hours. From a vomit of patients, from the market vendor's ice-cream and from the American ice-cream *Staph. aureus* type 17 was recovered. All three ice-creams were grossly contaminated and contained coagulase-positive staphylococci in enormous numbers. Four Army orderlies who prepared the ice-cream mix were investigated, but type 17 staphylococci were not isolated from the throat or nose of any of them. Others also handled the utensils, etc., but were not available for examination.

The authors emphasise the need to freeze any ice-cream mix almost immediately after it is prepared and this is now a requirement of the Ministry of Health for every manufacturer of ice-cream in which dried egg is incorporated.

To obtain a licence he must agree not to keep the mix at atmospheric temperature for more than 1 hour before pasteurisation, to subject the mix to heat treatment comprising exposure to a temperature of not less than 165°F. for at least 30 minutes, and to cool the mix down to 40°F. within $\frac{1}{2}$ hour after pasteurisation.

Synthetic Cream

Hobbs (1951) points out that the dangers arising from the contamination of synthetic cream are far greater than ice-cream,

which is kept frozen and handled comparatively little, and remarks:

Pasteurisation of the mixed ingredients is probably carried out by large manufacturers only. There must be a vast number of small bakeries where the cream is made-up and handled with scant attention to principles of hygiene.

OUTBREAKS OF INFECTIOUS DISEASE IN ENGLAND AND WALES CAUSED BY IMITATION CREAM

Date	*Place*	*Disease*	*Infective Bacterium*
1942	Bristol	Paratyphoid fever	*Salm. paratyphi* B
1944	Epsom	Food poisoning	*Paracolon bacilli*
1946	London	Food poisoning	*Salm. typhi-murium*
1946	London	Food poisoning	*Salm. thompson*
1948	Lincoln	Food poisoning	*Salm. typhi-murium*
1948	Eastbourne	Paratyphoid fever	*Salm. paratyphi* B
1948	Lambeth	Food poisoning	*Salm. typhi-murium*
1949	London	Food poisoning	*Salm. typhi-murium*
1949	Llanelly	Food poisoning	*Salm. typhi-murium*
1949	Newcastle	Food poisoning	*Salm. typhi-murium*
1949	Bolton	Food poisoning	*Salm. typhi-murium*
1950	London	Food poisoning	*Salm. typhi-murium*
1950	Cardiff	Food poisoning	*Salm. typhi-murium*
1950	Frimley and Camberley	Food poisoning	*Salm. typhi-murium*
1950	Exeter	Food poisoning	*Salm. typhi-murium*
1950	London	Paratyphoid fever	*Salm. paratyphi* B
1951	Wolverhampton	Food poisoning	*Bact. coli*
1951	Sunderland	Paratyphoid fever	*Salm. paratyphi* B
1951	Birmingham	Paratyphoid fever	*Salm. paratyphi* B
1951	Portsmouth	Paratyphoid fever	*Salm. paratyphi* B
1952	Whitby	Paratyphoid fever	*Salm. paratyphi* B
1952	London	Food poisoning	*Salm. typhi-murium*
1952	Cardiff	Food poisoning	*Salm. typhi-murium*
1952	Manchester	Paratyphoid fever	*Salm. paratyphi* B
1952	Cardiff	Paratyphoid fever	*Salm. paratyphi* B
1952	Swansea	Paratyphoid fever	*Salm. paratyphi* B
1953	Stafford	Food poisoning	*Salm. typhi-murium*
1953	London	Food poisoning	*Salm. typhi-murium*
1954	Cardiff	Paratyphoid fever	*Salm. paratyphi* B
1954	London	Paratyphoid fever	*Salm. paratyphi* B
1954	London	Paratyphoid fever	*Salm. paratyphi* B
1954	London	Paratyphoid fever	*Salm. paratyphi* B
1954	London	Paratyphoid fever	*Salm. paratyphi* B
1954	Windsor	Paratyphoid fever	*Salm. paratyphi* B
1954	Basingstoke	Paratyphoid fever	*Salm. paratyphi* B
1955	Worthing	Paratyphoid fever	*Salm. paratyphi* B
1955	Weymouth	Paratyphoid fever	*Salm. paratyphi* B
1955	Manchester	Paratyphoid fever	*Salm. paratyphi* B
1956	London	Paratyphoid fever	*Salm. paratyphi* B
1956	Bexley Heath	Paratyphoid fever	*Salm. paratyphi* B

Kwantes (1952) records an explosive outbreak of food poisoning, caused by *Salm. typhi-murium*, which affected some hundreds

of persons in Llanelly following the consumption of 'synthetic cream' pastries. The organism was isolated from the faeces of 169 cases and 94 symptomless excretors.

Specimens of faeces were requested from eight members working in the bakery and six of them were found to contain *Salm. typhi-murium*. One of the excreters had taken home some of the cream pastries on the Saturday, both she and her sister ate them for tea and had symptoms of infection the following day. The remaining five were symptomless excreters, two of whom were concerned in making the pastries. The possibility exists that one of these two infected the pastries. There is another possibility, namely that the cream mix was infected by the egg albumen. Both hens' and ducks' eggs were received by the establishment and duck egg albumen infected with *Salm. typhi-murium* may have been used in the synthetic cream mixture.

Experiments were made by Hobbs and Smith (1954), on the growth of *Salmonella paratyphi* B, coagulase-positive staphylococci and coliform bacilli, in synthetic cream prepared commercially or in the laboratory. The experiments showed that the growth of the test organisms could be suppressed by the addition of small amounts of hydrogen peroxide. It was therefore suggested that the spread of intestinal disease by this particularly vulnerable foodstuff might be controlled by the addition of hydrogen peroxide during manufacture.

Legislation for Cream Substitutes:

Food and Drugs Act, 1955, Part II, Sections 47 and 48.

Food and Drugs (Milk, Dairies and Artificial Cream) Act, 1950, Part III, Sections 29, 30, and 31.

Duck Eggs

There appears to be no practicable method of preventing with certainty the occurrence of salmonella infection in ducks, though their exclusion from access to human or animal excreta and other likely sources of infection doubtless would diminish its frequency.

With regard to the prevention of illness from the consumption of infected duck eggs, soaking the dirty ones and washing them with hypochlorite solution followed by brushing and water rinsings, has been found unsatisfactory. Brushing tends to force the pathogenic organisms through the shell pores. Boiling the duck eggs until the yolk is quite hard (15 minutes) is the only real safeguard. Ordinary frying, which leaves the yolk only partially cooked, cannot be considered safe. Any article of food containing duck eggs must be thoroughly cooked.

Scott (1943) gives an extract from an article by Sieke (1943) on the temperature attained in cooking ducks' eggs:

> The author has determined the temperatures attained by cooking for 8 minutes and for 10 minutes, registering it near the shell and deep in the middle of the yolk. He used eggs of an average weight of 75·4 gm., of length 6·3 cm., breadth 4·7 cm., and shell thickness 0·38 mm. Six eggs were used for each experiment. Since it is the custom in many households to cool down the egg with cold water after boiling, estimations were made before and after such cooling down.
>
> (1) *Eight Minutes' Boiling.*—The temperature in the middle of the yolk rose from 20°–30°C. at the start of boiling (according to the size and weight of the egg) to 65°–74°. Heat was slowly transmitted from the outer layers, and a higher temperature of 76°–78° was reached after removal of the eggs from the boiling water. The cooling 'down' reduced this by one degree. This cooling down had great effect on the cooling time in the interior. Without it the temperature of the yolk remained over 60° for 25 minutes and over 70° for 12–15 minutes, whereas these temperatures were registered after cooling down for only 10–13 and 5–7 minutes respectively.
>
> (2) *Ten Minutes' Boiling.*—The temperature in the yolk rose to 74°–82°C.; and later, after removal from the boiling water to 89° or, if cooled down, to 82°–87°; it remained over 60° for 34–36 minutes and over 70° for 24–25 minutes, and over 80° for 13–14 minutes, if uncooled, but, if cooled down, the respective figures were, over 60° for 14–16 minutes, over 70° for 10–12 and over 80° for 3–7 minutes only.
>
> The temperature taken 1·1 cm. within the shell naturally showed a higher maximum reached, a more rapid fall after removal, and a much more rapid fall after 'cooling down'.
>
> When these temperatures were compared with those found necessary to kill paratyphoid organisms in broth culture or milk, namely 60°C. for an hour, while 70° for 25 minutes will not suffice, it is obvious that cooking of ducks' eggs for shorter periods than 8 minutes will certainly not render an infected egg harmless.

Winter, Stewart, McFarlane, and Solowey (1946) record the results of experiments made to discover the times and temperatures necessary (using a model pasteurizer) to destroy salmonella organisms suspended in liquid egg. They state:

> It has been found (Rettger, Hull and Sturges, 1916) that soft boiling, codling, or frying on one side did not always render an egg free from Salmonella organisms. Eggs artificially infected with *S. pullorum* (Tittsler, 1930) required a 5 minute boiling to kill all the organisms. Van Oïjen (1940) and Wedeman (1940) recommended that duck eggs be boiled 10 minutes before use or the broken out liquid heated at 149°F. for 20 minutes to destroy *S. enteritidis* and *S. paratyphi* which are sometimes present.
>
> Salmonella organisms were not difficult to destroy in the pasteurisation unit. *S. senftenberg* and *S. cerro* were the most resistant types and *S. pullorum* the least resistant. Strains of *S. senftenberg* which did not

darken Kliger's iron agar were more resistant than those which produced a darkening of the medium. With the two exceptions noted the Salmonella types (*S. pullorum, S. oranienburg, S. montevideo, S. tennessee, S. anatum, S. bareilly, S. typhi-murium, S. meleagridis, S. london, S. newington, S. derby, S. rubislaw, S. oregon, and S. kentucky*) were destroyed in liquid whole egg at 150°F., within 0·3 minutes, at 148°F. within 0·8 minutes, at 146°F. within 0·8 minutes, at 144°F. within 1·2 minutes, at 142°F. within 2·0 minutes, at 140°F. within 2·6 minutes, and at 138°F. within 3·7 minutes. [See also Wilkin and Winter (1947); Solowey, Sutton and Calesnick (1948).]

Cathcart, Merz, and Ryberg (1942) remark that:

> Merely bringing custard to a second boil, after the addition of the thickening mix, rendered them sterile of both *Staphylococcus aureus* and *Salmonella enteritidis* with which they had been inoculated. Baked custard pies, which had been inoculated with Salmonella enteritidis and Staphylococcus aureus before baking, were sterile in relation to these two organisms as they left the oven.

Clarenburg and Burger (1950) carried out experiments on the survival of salmonellae in boiled duck eggs. The observers point out that

> from a practical point of view, it is important that the temperature of the centre continues to rise after the boiling process, when the egg is cooled in the air or in cold water. If 10 minutes boiling is followed by immersion in water at 0°C. (32°F.) the central temperature still rises, 11°C. (52°F.) enough to kill the *Salmonellae*. Thus, strictly speaking the destruction of the bacilli is caused in this case by the rise in temperature after 'boiling' and during the 'cooling'. If, however, the egg is eaten at once after 10 minutes boiling, or its contents cooled down immediately by slicing or chopping, viable *Salmonellae* may be present and constitute a health hazard.

In 1954, members of a salmonella sub-committee of the Public Health Laboratory Service issued a report on the results of a survey made during the years 1950–2 on salmonella in duck eggs.

> A total of 13,562 ducks' eggs were examined singly or in small batches in eight laboratories in England. It is estimated that the infection rate of individual eggs was about 0·15 per cent. In 483 examinations, representing 1,263 eggs, from shops, only a single infection was detected, due to Salm. typhi-murium. In 3,934 examinations representing 12,299 eggs from packing stations, 26 isolations of Salmonellae—3 Salm. pullorum, 2 Salm. enteritidis and 21 Salm. typhi-murium—were made. Salmonella infection in ducks' eggs was present in 7 out of 421 farms examined in 1951, and 3 out of 169 farms in 1952.

Spray-Dried Egg

The consumption of spray-dried egg in this country is of considerable importance, owing to the fact that there was a marked

increase in the number of outbreaks of salmonella food poisoning, especially during the war years 1943-4. This increase was attributed partly to the consumption of infected spray-dried egg.

According to Medical Research Council (1947), Spec. Rep. Ser. No. 260:

American spray-dried egg was imported into Great Britain on a small scale towards the end of 1941. From then until July 1942, its distribution was restricted to bulk supplies to bakers, confectioners and caterers. In July 1942, the first issue of 5 oz. packets was made through retailers to the general public. A similar issue was continued at monthly intervals till January 1946. Nearly all the egg powder for retail supply came from the United States; of the bulk packages distributed to the bakery and catering trades, 75 per cent came from the United States and 25 per cent came from Canada and the Argentine.

Between the years 1943 and 1945, the Medical Research Council in conjunction with the Ministries of Food and Health arranged for the bacteriological examination of 7,584 samples of spray-dried egg from the United States, Canada, and the Argentine. The results showed that 9·9 per cent were infected with organisms belonging to 33 different species of the salmonella group. All the species are believed to be potentially pathogenic to man.

Several new species of salmonella were isolated and identified. Of these, the 6 commonest were *oranienburg*, *montevideo*, *meleagridis*, *anatum*, *tennessee*, and *bareilly*. Regarding new species, Haines and Wilson (1947) remark:

Since the new species of Salmonella were never met with in cases of food poisoning in this country before the introduction of American dried egg, and since both the time-incidence of the cases and the alteration in the species-distribution of the strains coincided very closely with the supply of dried egg to the human population, it is concluded that dried egg was probably responsible for a considerable proportion of the greatly increased number of outbreaks of food poisoning that occurred during the years 1943 and 1944.

During 1944-5 it was ascertained that spray-dried egg which was unfit for human consumption was being used as pig food without previous sterilization. The Medical Research Council, in conjunction with the Ministry of Food, then arranged for the examination (in the laboratories of the Emergency Public Health Laboratory Service) of the mesenteric lymph nodes from 5,285 pigs which had been killed in various parts of the country. As a result of this examination 133 strains of salmonella were isolated; an incidence of 2·5 per cent. Among these were: *montevideo*, *oranienburg*, *anatum*, *give*, *derby*, *panama*, *bovis morbificans*, *oregon*,

newington, *infantis*, and *norwich*. All but three of these new species are known to be present in spray-dried egg. It was concluded that the pigs had become infected in this way.

In the general summary and conclusions of the aforesaid special report No. 260 (1947), the Medical Research Council states:

> The epidemiological and bacteriological evidence submitted in this report leaves little doubt that the introduction into this country of American spray-dried egg led to a considerable increase in the amount of food poisoning and other forms of Salmonella infection in the human population, and to infection of some, at least, of our farm animals. That the amount of human infection was not greater than it was can be ascribed, we believe, to the educative and restrictive measures taken by the Ministry of Food to minimise the risk of infection.

Gibbons and Moore (1944) examined Canadian egg powder and isolated salmonella strains in 28 samples (7·4 per cent) all but 5 (*Salm. pullorum*) were potentially pathogenic to man. The source of infection was not definitely established, i.e. whether from the interior, the exterior, or by some other means in the process of preparation.

Solowey, Spaulding, and Goresline (1946) in an investigation of a source and mode of entry of salmonella organisms in spray-dried whole-egg powder record that:

> Microbiological examination of more than 5000 samples of spray-dried whole-egg powder (94 to 96 per cent total solids content) manufactured in the United States between September 1st, 1943, and January 1st, 1945, has revealed a relatively high incidence of *Salmonella* contamination. . . . In view of the nature of the dehydration process, it would seem that the *Salmonella* organisms enter the processing system by way of the raw material. If such is the case, they may be introduced into the egg at the time of its formation within the hen, or deposited on the shell at the time of laying or by later contact with faecal or soil material. Micro-organisms derived from faeces, manure and soil may penetrate through the shell and shell membrane and develop within the egg meat if temperature and humidity conditions are favourable. . . . The evidence obtained in these studies indicates that the external shell surface is an important source of Salmonella contamination and that dirty eggs are the primary offenders. These limited results do not, however, rule out the possibility of an internal yolk infection or the possibility that organisms in an infected yolk may migrate into the membrane and possibly into the shell. . . . The findings of a wide variety of *Salmonella* types on shell surfaces and in the ground shell adds support to the evidence that contaminated egg-shell surface is a significant source of the Salmonella organisms found in the finished powder. . . . The fact that 31 per cent of the newly made powder samples were positive is conclusive evidence that the dehydration process as commonly

practised in the manufacture of powder of 4 to 6 per cent mixture content is not markedly destructive to Salmonella organisms.

Solowey-McFarlane, Spaulding, and Chemerda (1947) record:

Incidence of Salmonella in samples examined from individual plants ranged from 0 to 71 per cent. Fifty-two Salmonella types were identified, Culturally, morphologically, biochemically and serologically, the 52 types are in every way similar to those isolated from human beings and animals.

Scott (1930) and Brown, Coombs, and Wright (1940) demonstrated the penetration of *Salm. aertrycke* through the egg shell. Rettger (1913), Schalm (1937), Haines and Moran) 1940), Mallmann and Davidson (1944), and others have demonstrated penetration of a variety of micro-organisms through the egg shell. Environmental conditions under which eggs are handled today on farms, in stores, in produce houses, in cold-storage houses, and at dehydration plants frequently are favourable for shell penetration.

Solowey, Rosenstadt, Spaulding, and Chemerda (1948) confirmed the occurrence of multiple salmonella types in spray-dried whole-egg powder. See also McCullough and Eisele (1951).

It is generally agreed that it is most essential that a high standard of cleanliness and general sanitation should be maintained in the factory where dried egg powder is prepared and adequate measures adopted to exclude unsound or contaminated eggs. No duck, turkey, goose or guinea-fowl eggs should be used in the production of dried egg powder.

Regarding the use of dried egg for domestic purposes, if this commodity 'is effectively cooked immediately after reconstitution, the risk of Salmonella infection does not arise' (Min. Food, 1947).

Egg-Albumin, Imported

Considerable quantities of egg-albumin in crystalline form have been imported into this country during the past few years. The product is used extensively in the bakery and confectionery trades. In July 1955, consignments were found to be heavily contaminated with salmonellae and occasionally by *Salm. paratyphi* B (Newel, Hobbs, and Gordon-Wallace, 1955). The contamination varied in different consignments, but in one instance it was 47·60 per cent.

In 1956, Meredith, Davies, and Parry carried out experiments to discover whether a form of heat treatment could be introduced to make the product safe for commercial use. In a preliminary

report they suggested a practical and effective method of heat-treating bulk supplies of egg-albumin crystals at 130°F. for $5\frac{1}{2}$ to 6 days. It was recommended that it be made obligatory for all imports of egg-albumin to be powdered and heat-treated on arrival in this country. It is understood that further experiments are being carried out to ascertain whether a $5\frac{1}{2}$-day treatment has a sufficient safety margin.

Salmonellosis in Ducklings, Ducks, Chickens, and their Eggs

In Germany infection of fowls with *Salm. typhi-murium* has been reported by Beller and Zeki (1934).

Lütje (1937) recorded infection of fowls with *Salm. dublin.*

Edwards (1939) states that poultry constitutes the greatest reservoir of paratyphoid infection in the United States and mentions that most of the types found in fowls are capable of producing disease in man. Edwards and Bruner (1943) record that 43 organisms of the salmonella group have been isolated from poultry in the United States.

In this country the infection of chicks and ducklings by salmonella organisms is not uncommon. According to Garside and Gordon (1940), and Gordon and Garside (1944), the only salmonella types recorded, apart from *Salm. pullorum* and *Salm. galinarum*, are *Salm. typhi-murium* and *Salm. enteritidis.*

Gordon and Buxton (1945) remark:

> During the years 1943 and 1944, Salm. thompson, not hitherto reported in poultry in this country, has been isolated on forty-four occasions from thirty-one outbreaks in chicks and two outbreaks in ducklings.

They describe in detail two extensive outbreaks in chicks due to *Salm. thompson* (this organism is now a common cause of food poisoning in this country), and discuss the epidemiology of the disease and its possible importance to public health. The authors of this paper have isolated the following additional types not previously reported in poultry in this country: *Salm. bareilly*, *Salm. california*, *Salm. montevideo*, and *Salm. anatum.*

Wilson (1944) described an outbreak of disease in chicks, due to a mixed infection with *Salm. aertrycke* and *Salm. thompson*, in which a possible source of *Salm. aertrycke* was dried milk powder contaminated with mouse faeces from which this organism was isolated. *Salm. thompson* was demonstrated in the intestines of mice caught in the brooder house, and it was considered that this represented a possible primary source of infection. In view of

more recent work, it now appears that although this surmise may have been correct, it is more probable that the mice acquired the disease through contact with infected chicks and disseminated rather than introduced the disease.

Wilson (1945) made further investigations into salmonella outbreaks among chicks and ducklings and describes the method of bacteriological examination of eggs.

Swabs were made by covering small 'balls' of cotton wool with square pieces of gauze, the ends being twisted and tied with thread to form a 'neck' for easy manipulation with forceps during the operations of swabbing.

After being sterilised, the swab was moistened with sterile broth, passed over the surface of the shell and dropped into a flask of tetrathionate broth. The egg shell was next washed in 5 per cent dettol solution, dried, plunged into methylated spirits, and then flamed. Dipping and flaming were repeated a second time. The tip of the shell was removed by breaking with sterile forceps. The albumen decanted and discarded and the yolk poured into a large tube of brilliant green peptone water. Shells were examined in groups of six, but yolks were incubated individually.

Platings were subsequently made on McConkey medium, suspicious colonies picked off and submitted to carbohydrate fermentation tests and to serological tests, using sera from the Standards Laboratory, Oxford. *Sal. thompson* was isolated from two lots of six shells from each farm. The yolks were negative in every case.

Wilson also suggests certain measures to control infection. The following is his summary and addendum:

(1) A serious outbreak of disease in young chicks giving rise to a mortality reaching up to 36 per cent due to infection with *S. thompson* and *S. aertrycke* is described.

(2) The isolation of *S. thompson* and *S. aertrycke* from the shells of hens' eggs and *S. thompson* from the shell of a duck's egg is recorded.

(3) Bacteriological examination of cloacal swabs has shown that faecal contamination is a probable source of this infection.

(4) The setting of contaminated eggs is shown to be a means of spreading the disease within the incubator, and that the wiping of dirty eggs or the handling of infected eggs may spread infection in others. Secondary infection takes place in the brooder.

(5) It seems possible that mice may become infected with *S. thompson* during contact with diseases chicks and may play a part in the further dissemination of infection through contamination of food supplies.

(6) 'Custom-sexing' on affected premises may be the means of setting up fresh outbreaks.

(7) The agglutination test is unlikely to be a practical or effective method of eradication.

(8) Disinfection of eggs within the incubator by formaldehyde gas is suggested as a control measure where the disease is suspected.

Fumigation of the chicks during hatching should also be practised. The spraying of the floor and lower walls of the incubator room during removal of chicks for packing or sexing may be a useful additional measure. Where hatching and rearing are carried out on a large scale the provision of duplicate brooder rooms is suggested.

(9) The presence of Salmonella on egg shells may be a method of infection in cases of food poisoning in man. It is possible that penetration of the shell may occur, resulting in infection of the contents and increasing the risk of food poisoning. Failure to isolate *S. thompson* or *S. aertrycke* from the yolks of the eggs examined is recorded, but it seems probable that such infections do occur.

The bacteriological examination of eggs from hens and ducks naturally infected with *S. thompson* has been continued, and the organism has been isolated in several cases from the yolk, albumen and the inside of the shell. This would appear to be the first time that a Salmonella other than *S. pullorum* has been recorded in hen's eggs in this country, and the first occasion on which *S. thompson* has been isolated from ducks' eggs. In view of these findings, the control measures suggested cannot be absolute, but probably still represent the most practicable method of control of the disease until such time as an efficient method of diagnosis of 'carriers' becomes available.

Gibbons and Moore (1946) carried out some experiments on artificially infected fowl as carriers of salmonella. They found that

When aqueous suspensions of *Salm. bareilly* from agar were fed to adult hens the organisms were eliminated from the intestinal tract in 2 to 4 days; when broth cultures were fed, they were excreted over periods of 18 to 38 days. In adult fowl the organisms apparently localise in the intestinal tract. It is therefore quite possible that eggs may be contaminated through faecal matter. In these experiments S. bareilly was isolated from the shells of 3 of 37 eggs laid while this organism was being excreted.

Buxton and Gordon (1947) state:

The results of blood-testing and bacteriological examination of cloacal swabs, random faecal samples and eggs indicated that:

(1) Many chicks which survived an outbreak of *S. thompson* continue to carry the organisms for some months without showing any symptoms. In most cases the organism was harboured in the intestines and was excreted intermittently in the faeces. On one occasion *S. thompson* infected the gall bladder and the bacilli were excreted in the faeces for at least 18 months after the outbreak had occurred.

(2) The common method of egg infection was by the contamination of the shell with infected faeces. Under conditions of incubation the bacilli penetrated the egg shell and infected the yolk. Although there was little penetration of the organism under storage conditions, the bacilli on infected shells remained viable for at least 21 days.

(3) The common methods of spreading infection in a hatchery were from:

(*a*) The contact of infected and non-infected egg shells.

(*b*) The handling of eggs before and during incubation.
(*c*) The contact of egg shells with infected incubators.
(*d*) The ingestion and inhalation of infected fluff and incubator debris at hatching time.
(*e*) The ingestion of food and water contaminated with infected faeces from survivor chicks.

For the control of *S. thompson* infection in poultry, the following procedures have been recommended:

1. The production of agglutinins by carrier birds was not a reliable indication of infection. In known infected flocks, however, the detection of carriers by blood testing and by the examination of cloacal swabs is of value. For such a test an alcholised antigen is preferable to a heat-treated broth antigen, and a titre of 1/20th or more should be regarded as an indication that the bird is infected.

2. Only clean eggs should be used for hatching. Dirty egg shells should be cleaned by scrubbing or brushing and not by wiping with a damp cloth.

3. Fertile eggs should be stored in a cool, dry atmosphere for as short a period as possible before incubation.

4. Eggs should be fumigated in the incubator with formaldehyde vapour, not later than 24 hours after they have been set. [See also Gordon (1950).]

Regarding the control of salmonellosis in poultry, with special reference to fumigation of incubators, Wilson (1951), as a result of his investigations and experiments, points out that:

Fumigation with formaldyhyde gas produced by the addition of 1·5 cc. of formaldyhyde to 1 grm. of potassium permanganate per cubic foot of incubator space (approximately 5 oz. formaldyhyde and 3 oz. potassium permanganate per 100 cubic feet) is invariably lethal for Salm. typhi-murium on egg shells and on fluff, and normally has no adverse effect on hatchability, providing certain precautions are taken. Where the disease persists, direct egg transmission is probable and a combined programme of agglutination testing and fumigation is necessary.

Frank and Wright (1955) report experiments, designed to sterilize the contents of egg incubators, in which cultures of salmonella strains on egg shells and string were exposed to formaldehyde fumigation in closed incubators. When 1 ml. formalin per cubic foot of incubator space was used, a period of 30 minutes was sufficient to kill all organisms. With 1·5 ml. per cubic foot 20 minutes was adequate.

With regard to salmonella infection from chickens' eggs, Watt (1945) records an outbreak which occurred on board an American merchant vessel in January 1945. In his summary he states:

An outbreak of salmonellosis (*S. montevideo*) aboard a merchant vessel, affecting 28 individuals in a crew of 70, is reported. Twenty-one

individuals were known to have had symptoms of varying severity, and from each *S. montevideo* was isolated. The same organism was isolated from seven additional members of the crew, none of whom reported any illness. Epidemiological evidence indicated that infection resulted from the consumption of contaminated egg salad, the mayonnaise of which contained raw eggs. The same Salmonella type, *S. montevideo*, was isolated from two cases of shell eggs obtained on the ship. Internal contamination of the eggs was demonstrated, since the shell washings before sterilisation were free of *S. montevideo*, and egg meats obtained after sterilisation of shells were found to contain this organism.

An outbreak of *Salm. typhi-murium* food poisoning, the vehicle for which was artificial butter and the source an infected hen's egg is described by Crowe (Dublin 1946).

It was characterised by a dramatic text-book onset and an almost familiar localisation. Twenty-three out of the twenty-seven persons at risk were affected and although the symptoms in some cases were very severe, all the patients eventually recovered. Although somewhat unusual, *Salm. typhi-murium* disease in human beings has, on a number of occasions, been caused by ducks' eggs. This particular outbreak is, however, of special interest as there is evidence to suggest that it was caused by a hen's egg, a source which, as far as I have been able to ascertain, has not been previously described.

Cantor and Macfarlane (1948) investigated salmonella organisms on and in chicken eggs. *Salm. montevideo* and *Salm. anatum* were isolated from 13 (0·6 per cent) of 2,132 samples of egg scrapings and strains of *Salm. pullorum* were isolated from 30 (1·2 per cent) of 2,584 egg meats. Salmonella organisms were not isolated from shell scrapings and meat of the same egg, although samples of both were available for 2,088 of the eggs examined.

A comparatively small number of surveys have been made in this country and abroad regarding the natural occurrence of salmonellosis in shell eggs. This is probably due to the large amount of experimental work which would be involved in the examination of a sufficient number of eggs. In one survey by Solowey, Spaulding, and Goresline (1946), 2 per cent of clean eggs and 16 per cent of dirty eggs were found to contain salmonellae on the surface or in the pores of the shells. Contamination of hen eggs with salmonellae, other than *Salm. pullorum*, was recorded by Wilson (1950) and by Carter, Powell, and Borte (1950). Wilson demonstrated the presence of *Salm. typhi-murium* in each of three eggs laid by a hen known to be infected.

In some instances the ovaries and oviduct (usually sterile) may be infected and the organisms deposited actually inside the egg, leading to extensive multiplication in the albumin and yolk. It

has been found, however, that contamination is much higher on or in the shells than internally.

Stokes, Osborne, and Boyne (1956) have shown that under suitable conditions of temperature, salmonellae in the shell can penetrate the shell membranes and multiply in enormous numbers within the egg. Moreover, the initial degree of contamination need not be very large for infection to occur. The greater danger in this mode of infection lies in the possibility that a single shell egg heavily infected with the organisms can contaminate large batches of sound eggs when broken out to produce liquid, frozen, or dried whole egg, albumin, or yolk. The data contained in the investigation suggested that shell eggs which are to be stored for several weeks or longer, should be kept below a temperature of 10°C. to prevent infection, penetration, and growth of salmonellae.

Murdock (1954) reports a survey made during 1951–3 in Northern Ireland where 1,010 batches of hens' eggs were examined at a factory where they were being processed into liquid egg. Salmonellae, usually *Salm. typhi-murium*, were isolated from 2·10 per cent of the eggs. Murdock (1956) points out that

> it would appear, therefore, that small numbers of hen eggs may be infected with Salmonellae pathogenic to man, and that these might contaminate large batches when the egg was being homogenized and mixed. This condition is borne out by the increasing evidence of the contamination of frozen and dried egg products with Salmonellae. [See Newell, Hobbs, and Wallace, 1955.]

Savage (1956) remarks:

> The danger does not arise from unopened shell eggs (except duck eggs from infected farms) but from pooled mixtures of shell eggs involving the distribution through the mass of any salmonellae present and usually also some multiplication of the bacilli. An important feature is the unknown (to health authorities) and insidious way these infected egg mixtures are distributed all over the country and used by bakers and confectioners in cakes and confectionery products which subsequently do not receive effective heat treatment.

Contamination of Food by Rats and Mice

Everything should be done to prevent the access of rats and mice to food destined for human consumption. This may be accomplished by the rat-proofing of buildings and stores and the storage of foods in rat-proof containers. Incidentally, food manufacturers, owners of warehouses and similar premises in which produce and supplies are subject to infestation by vermin, have found that the cost of proofing their buildings is in the long run the cheapest form

of insurance, and it is without doubt the greatest factor in the prevention of infection of food by rats and mice.

Strict attention should be given to the storage and disposal of all refuse and garbage. Water tanks and cisterns must be provided with proper fitting covers. In warehouses, especially where dried food is stored, the water supply should be cut off; this precaution often causes rats to leave the premises.

The measures for the prevention and eradication of rats and mice are well known and need not be described. Legislation: Prevention of Damage by Pests Act, 1949; Part I deals with Rats and Mice; Part II with Infestation of Food, and Part III is Supplemental.

Rat Viruses

A word, however, may be usefully added regarding the use of rat viruses. Savage and Bruce White examined a selection of these preparations and confirmed the view expressed by others in the past that these strains (e.g. Danysz, Liverpool Virus, Ratin) are typical enteritidis forms.

Jordan (1931) states:

> A real danger to public health undoubtedly resides in the employment of so-called 'rat viruses' for the extermination of these vermin. . . . Its use in kitchens and pantries may be the direct cause of food poisoning. There are on record a score or more instance where the careless use of a commercial rat virus has been followed by human infection, sometimes with fatalities. Since the method has not proved of material value in the destruction of rodents, and is, moreover, open to the serious sanitary objection that the animals after apparent recovery may continue to carry *Salmonella* bacilli and so contaminate food, the employment of rat viruses seems without justification.

Leslie (1942) made some interesting investigations into the principal viruses which are used for rat and mouse control in Great Britain, and gives a bacteriological classification of the cultures contained in these preparations. His summary is as follows:

> (1) The six 'viruses', Liverpool, Danysz, London, Ready Rat Relief, Institut Pasteur and Ratin, which are the principal bacterial cultures at present employed for anti-rodent control in Great Britain, have been examined.
>
> (2) By means of reciprocal absorption tests all these six strains were found to be serologically identical with *S. enteritidis* Gaertner, antigenic structure, IX: gom:
>
> (3) From the results of the fermentation tests, which may be used to subdivide this serological type, Liverpool, Danysz, Ready Rat

Relief and Ratin were assigned to the var. Danysz subgroup; while the London and Institut Pasteur strains could not be distinguished from the classic *S. enteritidis* type.

(4) Both of these subgroups are pathogenic for man, and evidence is cited which shows quite clearly that human cases of gastro-enteritis have been caused by the use of virus preparations. There are, also, reasonable grounds for believing that these bacterial types may be pathogenic for a number of domestic animals, including some poultry.

In a second report by the working party (convened by the Ministry of Agriculture, Ministry of Food, Ministry of Health, and the Scottish Office) on precautionary measures against toxic chemicals (Toxic Chemicals in Agriculture, Residues in Food 1953) the subject of bacterial rodenticides is reviewed and safeguards suggested in their use. Although these preparations are not on sale to the general public, they have been used by certain firms on a considerable scale. The recommendations include that the bacterial rodenticides should be limited to those containing *danysz* variety alone of *Salm. enteriditis* and used only by skilled operators of recognized firms. Also that these preparations should not be placed in food kitchens or other premises in which food is prepared or sold and that any unconsumed bait containing bacterial cultures be removed.

Taylor (1956) remarks:

> In this country between 1,944 and June 1955 Salm. enteriditis, var. jena, accounted for 1267 known cases of infection of which 21 were fatal, and Salm. enteriditis, var. danysz for 413 cases, of which 2 were fatal. In animals Salm. enteriditis, var. jena was isolated most commonly from guinea pigs, mice and pig glands, and Salm. enteriditis, var. danysz from rats, guinea pigs and mice. The use of bacterial rodenticides containing either of these organisms is no longer necessary and should be stopped.

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Chapter VIII

CLOSTRIDIUM WELCHII FOOD POISONING

THIS organism, which was first isolated and described by Welch and Nuttall in 1892, has been suspected for some years as being a possible cause of food poisoning. It was recovered in 1895 from the faeces of persons involved in outbreaks described by Klein (1895) and Andrews (1899). McClung (1945) recorded four outbreaks in which *Cl. welchii* was isolated from the incriminated food. A volunteer who afterwards consumed some of the contaminated food also developed typical symptoms of food poisoning.

The powers of resistance to heat of *Cl. welchii* varies from 1 minute at 98°C. (Dunham, 1897), $1\frac{1}{2}$ hours at 100°C. (Rodella, 1910, Von Hibler, 1906), to 1–4 hours at 100°C. (Zeissler and Rassfeld-Sternberg, 1949). Headlee (1931) reported that spores were killed in 30 minutes at 90°C. and 5 minutes or less at 100°C. Oakley and Warrack (1953) have shown that the heat-resistant strains only produce small amounts of toxins and suggest that these strains should be classified as *Cl. welchii*, type A. The strains are frequently isolated from normal faeces and certain foodstuffs.

The incubation period ranges from 8 to 12 hours (average 10 to 12 hours) and the symptoms are nausea, abdominal cramps, and diarrhoea usually without vomiting. There is no prostration or pyrexia, headache is rare, and recovery rapid. During the Second World War several outbreaks occurred among school children whose dinners were prepared in a communal kitchen (Knox and Macdonald, 1943).

Hobbs, Smith, Oakley, Warrack, and Cruickshank (1953) made a special study of food poisoning caused by *Cl. welchii*. In their summary they state:

(1) Outbreaks of mild food poisoning have been investigated in which heat-resistant Cl. welchii appeared to be the causative organism. The outbreaks were characterised by colic and diarrhoea without vomiting, commencing 8–20 hours after ingestion of the contaminated food.

(2) The strains of Cl. welchii concerned are only feebly toxigenic, and apart from heat-resistance of their spores, and some colonial characters, fit well into Cl. welchii Type A. The toxin production and the serology of the strains in uniform within an outbreak.

(3) Mild food poisoning similar to that seen in natural epidemics has been produced in volunteers by ingestion of cultures of heat-resistant Cl. welchii isolated from contaminated meat.

(4) Infection is almost invariably due to meat which has been boiled, steamed, braised, stewed or insufficiently roasted, allowed to cool slowly and eaten the next day, either cold or reheated.

In an experimental study of animal faeces, *Cl. welchii* was rarely recovered from bovines, but commonly obtained from mice, pigs, and rats. The organism was also recovered from fresh meat, except lamb, and from blow flies.

The observers remark:

> Meat may be contaminated with Cl. welchii in various ways—by organisms in dust, or by faecal organisms of human or animal origin, and it is clear that this contamination can occur in slaughter-houses, in transit and in canteens. To prevent the transfer of Cl. welchii from the human intestines to foodstuff via the hands depends, as usual, on the personal hygiene of the food handler. [See also Beck, Foxall, and Turner (1954), Goudie and Duncan (1956).]

Dische and Elek (1956) described feeding experiments on human volunteers with heat-resistant *Cl. welchii* type A cultures from a strain which originally caused a food-poisoning outbreak. The infected food produced in the volunteers a short attack of diarrhoea and abdominal pain after an incubation period of 12 hours, identical with those symptoms occurring in food-poisoning outbreaks. After the feeding tests heat-resistant *Cl. welchii* spores were found in the stools of 97 per cent of the volunteers.

The danger of pre-cooking meat foods and allowing them to cool slowly at room temperature has been stressed frequently. If the procedure is unavoidable, arrangements should be made to ensure that the food, after pre-cooking, is rapidly cooled and afterwards placed in a refrigerator until required.

The number of known incidents due to *Cl. welchii* in England and Wales were: in 1953, 25; in 1954, 48; in 1955, 90; in 1956, 77; in 1957, 93 (compiled from 'Annual Reports on Food Poisoning' contained in *Mon. Bull. Minist. Hlth. Lab. Serv.*).

The Proteus Group

The organisms of this group, which can be readily destroyed by heat or disinfectants, are widely distributed in nature and present in decaying meat and other decomposing organic material of animal origin. They are frequently isolated from the faeces of man and animals. The common species is *Proteus vulgaris*.

Outbreaks of food poisoning of gastro-intestinal type have been ascribed to proteus by Levy (1894), Wesenberg (1898), Glucksmann (1899), Schumburg (1902), Ohlmacher (1902), Pergola (1910), Mandel (1912), Wichels and Barner (1925), Plahn (1937), Cooper, Davies, and Wiseman (1941), and others.

Bengston (1919), Savage (1920), and Tanner (1933) carefully studied many of the recorded outbreaks of food poisoning regarded as being due to proteus and reported that in none of these was it established that this organism was aetiologically concerned.

Jordan and Burrows (1935) over a period of 10 years prepared broth filtrates of proteus strains, many of which were regarded as the cause of outbreaks of food poisoning, and fed them to human volunteers without any observable effects.

Dolman (1943), in his summary on bacterial food poisoning, says:

Filtrates prepared from several strains of Proteus vulgaris, B. coli, gram-positive sporulating bacilli, and streptococci, were taken by numerous persons in amounts up to 50 c.c., without harmful effects. Meat pies were inoculated with cultures of some of these organisms and also eaten without ill effects, although their bacterial counts greatly exceeded those found in the foodstuffs from which the cultures were first isolated.

The following is quoted from an editorial article in *Public Health* (1941):

The key to the discrepancy is given by the laboratory studies (referred to) which show that only rare and special strains of B. proteus (or B. coli) have acquired the property of producing an enterotoxin. Given such a strain it may be accepted that B. proteus, like the special staphylococci, may be responsible for an attack of food poisoning. Our present knowledge, however, is to the effect that this property is much rarer with proteus strains than with staphylococci. This infrequence makes any outbreak proved to be due to B. proteus of special interest. Such a one has recently been reported by Cooper, Davies and Wiseman at Bristol. The outbreak was from brawn, the pigs' heads from which it was made being stored for three days in a brine bath, then sold to a canteen and only two days later made into brawn. The material was said to have been boiled for 3 hours. The actual number of cases is not stated, but nine persons were definitely affected, while there was an additional number of mild cases. The incubation period was 3 to 5 hours and the usual food poisoning symptoms of vomiting, diarrhoea and abdominal pain were present, but in 5 cases there were marked collapse and cyanosis; all patients recovered. Proteus strains were isolated from the brawn and from a number of samples of faeces. Several strains from both brawn and faeces were positive when tested for enterotoxin by the intraperitoneal kitten test. The brine pickle

also yielded similar strains of Proteus vulgaris. This outbreak can be accepted as due to a Proteus vulgaris which produced enterotoxin and which was allowed to grow for a number of days in the pigs' heads before being made into brawn. Either the boiling was not boiling or there was re-infection of the made brawn, for this organism has no high resistance to heat.

Philippi, Raffo, Vaccaro, Perez, Mercedes, and Carbezas (1948) record an outbreak of food poisoning in a family (father, mother, and 3 children) caused by the consumption of fried fish. The only organism of possible significance isolated from the faeces of the patients was *Proteus vulgaris*. The same organism was present in large numbers in the cooked fish. Agglutination tests were made with sera of the victims and of a number of normal persons. Kittens were also inoculated with culture filtrates of the proteus strains. From the results of these tests it was concluded that this organism was responsible for the outbreak.

From the evidence available it would appear that special strains of the proteus group which cause gastro-intestinal symptoms, are rare, and consequently cases of food poisoning ascribed to them are infrequent.

The Dysentery Group

The ingestion of food infected by members of the dysentery group, *Sh. sonnei* and *Sh. flexneri*, at times produce the typical gastro-intestinal disturbance seen in cases of food poisoning. Scott (1938) mentions an explosive outbreak which occurred in Holborn, London. About 20 persons were infected with *Sh. sonnei* after partaking of pease-pudding, the latter having been contaminated by a child convalescing from the disease. See also Clayton and Hunter (1928), Sowden (1933), Thompson (1938), Fyfe (1938), Bowes (1938), and Gorman (1950).

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PART II

Chapter IX

STAPHYLOCOCCUS FOOD POISONING

Historical

The earliest researches into staphylococcal food poisoning were carried out by Denys (1894). He investigated an outbreak (with one death) caused by the consumption of meat from a cow which had died from puerperal fever. Bacteriological examination of the incriminated meat and also the spleen from the fatal case revealed yellowish micrococci. Denys was, however, unable to infect laboratory animals with cultures made from the micrococci isolated. He stated, 'The harmful qualities of the meat from the cow that died of puerperal fever are quite probably due to the presence of *staphylococcus pyogenes*'.

Owen (1907) accepted the view put forward by Denys that micrococci were a cause of meat poisoning. Later, he investigated an outbreak among 19 persons due to the ingestion of dried beef and described the characteristic symptoms. Owen obtained a pure culture of white micrococci from the meat and considered these organisms to be the cause of the outbreak.

In 1914, Barber isolated a white micrococcus (*Staphylococcus albus*) from a cow's milk, the consumption of which caused acute attacks of gastro-enteritis. A significant fact was that freshly obtained milk from the cow proved harmless when consumed and only developed the toxic properties after incubation for some hours at room temperature. Barber inoculated sterile milk with a culture of the micrococcus isolated and incubated it for 8½ hours at 36°C. He afterwards drank 55 c.c. of the milk: gastro-enteritis occurred within 2 hours. This was apparently the first indication that a poisonous substance produced by the micrococci and not the micrococci themselves might be the cause of gastro-enteritis. It was, however, some considerable time before the valuable investigations and experiments carried out by Denys, Owen, and Barber were fully recognized.

Research and experimental work carried out in the United States by Dack and his colleagues (1930), Jordan (1930–1), Jordan and Hall (1931), Jordan, Dack, and Woolpert (1931), Jordan and

Burrows (1933–5), Stritar and Jordan (1935), and Chapman, Lieb, and Curcio (1937); by Minett (1938) in this country and by Dolman and his co-workers (1934–43) in Canada, showed that certain specialized strains of staphylococcus produced a noxious heat-resistant substance (enterotoxin) which causes food poisoning outbreaks.

The term 'enterotoxin' was adopted because the toxic substance so produced seems to exert its most conspicuous effects upon the gastro-intestinal canal or enteron (Dolman and Wilson, 1938).

Jordan (1931) says:

> It seems quite reasonable to suppose that a certain proportion of the recorded outbreaks of food poisoning of undetermined origin were caused by the toxic products of staphylococci, whose presence in the implicated food went undetected, and whose significance would not indeed until quite recently have been recognised. It seems also a reasonable conjecture that some other species of bacteria are likewise able to produce substances toxic for man when swallowed. Possibly a large proportion of the outbreaks of food poisoning of hitherto undermined nature may be cleared up by studies directed along this line.

The Chief Medical Officer of the Ministry of Health, in his Annual Report for 1936, in recording toxic outbreaks, says:

> Of the remainder, 26 were regarded, from the clinical course (very acute gastritis and enteritis within 3 hours of a suspected meal, followed by rapid recovery), as due to a 'toxin' already elaborated in the food as a result of bacterial growth and not due to Salmonella infection. From the suspected food, cultures of staphylococci, usually aureus, were isolated in 10 instances in such numbers as to suggest that they had produced the gastro-intestinal irritant responsible for the symptoms. In one instance (canned tomatoes) heat-resistant streptococci of the faecalis type (enterococci) were present in great abundance and almost pure culture.

There is no reliable evidence why only some strains of staphylococcus produce enterotoxins, or the conditions which induce them to develop this property. The problem of distinguishing enterotoxigenic from non-enterotoxigenic staphylococci remains unsolved. At present, no biochemical, agglutination, or phage-typing furnishes satisfactory criteria for their differentiation. There is, however, a concensus of opinion that most of the outbreaks of this type of food poisoning are caused by pathogenic coagulase-positive, pigmented and haemolytic staphylococci. Coagulase-positive strains are generally accepted as indicative of their pathogenicity. The enterotoxin, which has both pathogenic and antigenic properties, is somewhat resistant to heat and may not be completely destroyed by exposure for 30 minutes to 100°C.

Savage (1941) remarks:

> During recent years a moderate number of outbreaks of food poisoning have been shown to be caused by certain staphylococci which produce an endotoxin pathogenic to man; as the favourite vehicle is cream cakes and sometimes milk, it is possible that the milk is not only the vehicle, but that the organisms may be derived from an udder infection. This possibility cannot therefore be ignored.

An increasing number of staphylococcus food poisoning outbreaks have occurred from time to time in this country, but they have been comparatively few in comparison with those due to the salmonella group of organisms. Doubtless many mild cases occur which are not heard of.

In England and Wales the total number of incidents due to staphylococcal food poisoning notified during the past 5 years, were as follows:

1953	1954	1955	1956	1957
132	127	138	131	128

Compiled from Annual Reports on Food Poisoning contained in *Mon. Bull. Minist. Hlth. Lab. Serv.*

There is no doubt that staphylococcus food poisoning is of great importance but its incidence, however, appears to be much greater in the United States and Canada. Dack (1943) remarks:

> The long delay in recognition of the rôle of this organism in food poisoning may be attributed chiefly to the immense amount of publicity given Salmonella or paratyphoid organisms in food poisoning.

The following figures show the number of outbreaks and cases of staphylococcus food poisoning recorded in the United States Public Health Reports (Public Health Service) for the past 5 years:

Year	*Outbreaks*	*Cases*
1952	77	3,798
1953	81	4,045
1954	100	4,868
1955	102	4,130
1956	111	4,313

It is of interest to quote what Dolman (1943) has to say on the incidence of staphylococcal food poisoning:

> Its actual incidence is unknown, since the nature of the syndrome is such that outbreaks are only likely to be reported when fairly large groups are involved; while many health authorities, particularly in Great Britain, have been either unfamiliar with the conditions or sceptical of its existence. But the ubiquity of staphylococci, and the lack of any evidence pointing to a specially limited distribution of enterotoxigenic strains; the great variety of foodstuffs in which the enterotoxin is known to have been elaborated; and the prevailing

ignorance and apathy respecting the prevention of air, milk, finger, droplet, or fly-borne contamination of foodstuffs, make a high incidence of staphylococcal food poisoning inevitable. That the incidence is not still higher may be explicable in terms of recent findings in these laboratories, which indicate that among staphylococci isolated from various sources, food poisoning strains may not comprise as high a percentage as other workers have claimed.

Staphylococci organisms are commonly found in nature. They are present in the air, on eating and drinking utensils, human throats and nasal passages, boils, pimples, carbuncles, on the human skin and mucous membrane, as well as in localized abscesses, periostitis, septicaemica, pyaemia, urinary sepsis, wound suppuration, etc. This emphasizes that only certain strains can cause food poisoning. Recent surveys have shown that 50 per cent of normal adults harbour staphylococci in the nasal passages and in some 10–20 per cent they are present on the hands. *Staphylococcus aureus* is usually found in pyogenic lesions, but *Staphylococcus albus* strains sometimes prove to be pathogenic. The *aureus* type, however, is the most virulent.

Several workers have reported the association of the carriage of *Staphylococcus aureus* on the skin of the back of the wrist with its carriage in the nose. Gillespie *et al.* (1939) demonstrated the serological identity of the nose and skin strains; the numerical association was confirmed by Miles *et al.* (1944). These investigators also found that the incidence of nasal-carrier rate among people of the working class was between 19 and 65 per cent, with an average of 47·4 per cent. It has now been possible to extend the study by the use of the phage-type filtrates developed by Wilson and Atkinson (1945). See also Williams, Rippon, and Dowsett (1953).

Williams (1946) states:

> Bacteriophage typing has been used to study the association of nose and skin carriage of staphylococcus aureus. The coccus was isolated from the hand and nose of 65 patients; both strains were typable in 36 cases and were of the same type in 31, a proportion considerably in excess of that to be expected of a change basis. . . . Data from 22 subjects whose nose and skin were swabbed on eight consecutive weeks suggest that some of those people who were tested on one occasion only yield staphylococcus aureus from the skin but not from the nose, are intermittent nasal carriers, and that their skin is probably contaminated from the nose. . . . wrist carriage of staphylococcus aureus, like nose carriage may often be persistent over a number of weeks and the type carried is usually constant.

The results of experimental research support the conclusion that staphylococci are among the more heat-resisting of the

non-sporing group of organisms. Many of these organisms can withstand a temperature of 60°C. (140°F.) for half an hour. This power of heat resistance doubtless varies with the particular strain of staphylococcus. Beamer and Tanner (1939) reported *Staphylococcus aureus* survived 13·8 minutes and was destroyed in 18·8 minutes at 65°C. (149°F.). Jensen (1945) states:

Staphylococci are killed in pasteurising temperatures—61·7°C. (143°F.)—for 30 minutes. The *Staphylococcus* does not form poisons unless it grows and it only grows well at temperatures from 21·1° to 40·6°C. (70° to 105°F.). It requires 4 to 8 hours of steady growth (logarithmic phase) in a rich food medium at 37·2°C. (99°F.) to produce enough toxin to elicit symptoms (vomiting and diarrhoea) in man or monkeys when the food or broth is swallowed.

Wilson (1939 and 1942) describes a series of outbreaks of food poisoning caused by naso-pharyngeal human carriers.

A considerable amount of research has been carried out of late years by various investigators, mostly in the United States of America and Canada, into the production and detection of the enterotoxin and its effect upon human volunteers and on a number of the lower animals, under experimental conditions (Jordan and McBroom, 1931). Jordan (1930), in a series of tests (using filtrates of proved sterility) on a number of human volunteers, found that a considerable number of the staphylococcus strains examined possessed toxigenic properties. In these experiments strains were used from normal human throats, from a case of septicaemia, and from food incriminated in food poisoning outbreaks.

Filtrates from 48-hour cultures were found as toxic as those from 7-day cultures. Positive results were usually obtained when 5 c.c. amounts were swallowed, and the symptoms were often quite severe. Recovery was always prompt and complete.

Regarding the production of staphylococcus enterotoxin Gross and Vinton (1947) quoting Dack remark:

The comments of Dack (1943) are very interesting and pertinent, namely, that enterotoxin production is a function of growth and that enterotoxin could be demonstrated in cultures after 12 hours at 37°C. (98·6°F.) and three days at 18°C. (64·4°F.) but not in cultures grown at shorter periods or for 7 and 3 days at 9°C. (48·2°F.) and 15°C. (59°F.) respectively, or after four weeks at 4°C. (39·2°F.) and 6·7°C. (44·1°F.). Also important are the comments of Dack that certain foods such as canned salmon, may support growth without production of enterotoxin and that meat products allow food-poisoning staphylococci to grow well and produce enterotoxin.

The following remarks by Tanner (1933) on the toxic agent

secreted by the staphylococci causing food-poisoning outbreaks are interesting:

The difficulties experienced in studying the agent have been partially overcome according to Jordan and Burrows by the methods of administering the materials to monkeys as described by Jordan and McBroom. Using this technique Jordan and Burrows reported the following characteristics for the agent:

(1) The active principle will not distill.
(2) It is not readily dialysable.
(3) It is markedly unstable to N/100 NaOH.
(4) It is unstable to heat in N/100 HCl solution.
(5) It is not identical with the haemolytic substance present in many staphylococcus filtrates, nor does it produce a skin reaction.
(6) It is completely removed from acid aqueous solution by extraction with ethyl ether or chloroform as judged by our method of assay.
(7) It may be extracted from alkaline solution with ethyl ether or chloroform but the deleterious effect of alkali tends to mask such removal. Intravenous injection of the same solution into rabbits, cats and dogs produced no ill effects.

Davison, Dack, and Cary (1938) found that the typical symptoms followed the intravenous injection of rhesus monkeys with boiled filtrates from cultures of food-poisoning staphylococci in doses of 1 ml. (and at times even 0·2 ml.) per kilogram of bodyweight.

In these various researches special methods and media are necessary to ensure the growth of the staphylococci and the production of the enterotoxin. In testing the enterotoxin, cultures or filtrates were fed to the experimental animals or injections made of boiled cultures or filtrates into the body or blood-stream of the experimental animals.

In experiments carried out by Dolman, Wilson, and Cockcroft (1936) use was made of the kitten (or cat) as the test animal for staphylococcus enterotoxin. They explain that 'the cat is more akin to human beings than are rodents in dietary and excretory habits; is less prone to vomit than the dog; and is a more convenient and economical experimental animal than the monkey.' Later, other investigators included Davison, Dack, and Cary (1938) and Hammon (1941).

Dolman and Wilson (1938) state that staphylococcal enterotoxin may be detected by the intra-abdominal injection into kittens of filtrates whose alpha and beta toxins have been destroyed by heat or formalinization, or have been removed by preliminary absorption with serum containing anti-bodies to the alpha and beta toxins but not to the enterotoxin.

It may be mentioned that Tanner and Ramsey (1932) reported indifferent success with kittens. See also Matheson, Thatcher, and Simon (1955), 'Studies with Staphylococcal Toxins'.

Bayliss (1940) made an extensive study of the pharmacological action of the enterotoxin and tested its action on young and adult cats. These experiments showed that vomiting resulted from intravenous, intracardial, and intraperitoneal infections but failed to appear subsequent to oral, subcutaneous, or intramuscular administration. He concluded that the action of staphylococcal enterotoxin on peripheral sensory structures is of greater importance in the iniation of vomiting than the direct action of the toxins on the vomiting centre.

Savage (1943), in an abstract from an article by Fulton (1943) on 'Staphylococcal Enterotoxin—with Special Reference to the Kitten Test', says:

> A kitten-positive extract was non-toxic by mouth to a susceptible human volunteer and a kitten-negative extract was toxic to this volunteer.
>
> Of four strains of Staphylococcus aureus isolated from four food poisoning outbreaks three were negative by the intraperitoneal kitten test, one positive. The three negative strains produced only alpha lysins, the positive strain produced in addition a potent beta lysin. Unheated extracts of one of the negative strains (Wood 46) containing alpha but no beta lysin, injected intraperitoneally into a kitten caused vomiting within about 10 minutes, extreme collapse and death overnight; after boiling the extract for 20 minutes a similar injection caused no symptoms. Strain C 13344, containing large amounts of both alpha and beta lysins, both unheated and after boiling for 20 minutes, gave positive reactions in a kitten. The boiling reduced the beta lysin from 1/5000 to 1/256.
>
> Further experiments showed that kitten-positive strains produced potent beta lysins, whereas the negative strains did not. Steps are described for the purification of these lysins. Purified alpha and beta lysins injected intraperitoneally into kittens induced vomiting. Boiling for 20 minutes extracts which contained alpha and beta lysins destroyed most of the alpha lysins, leaving insufficient to induce vomiting, but there was enough beta lysin to cause the kitten to vomit. Views as to the heat lability of the alpha lysin must be revised, as in fact less activation occurs by boiling for a given time than by heat at 65°C.
>
> With a susceptible human volunteer, feeding experiments with suitable sterile filtrates showed that neither a strain with an alpha lysin but no beta (Wood 46) nor a strain (C 13344) with both lysins was toxic by mouth, but one (Jordan 7), producing alpha lysin (1/160 titre) and beta lysin with only a titre of 1/32, produced vomiting symptoms. In a toxic extract it was not found possible to separate the enterotoxin from the alpha lysin of that extract, and it cannot be said whether enterotoxin is distinct from or identical with the alpha lysin. The kitten

test is unreliable in the identification of the enterotoxin-producing strains.

Allison (1952) remarks:

Further investigations carried out by Fulton (Oxford) during the war years seemed to cast some doubt on the specificity of the test and is now little used in this country. Fulton examined at Oxford the bacterial flora of suspected foods from 35 outbreaks of food poisoning during the war years. In one outbreak the food contained large numbers of coagulase-positive staphylococci, in 5 outbreaks *Proteus vulgaris* were present, and in 3 the two organisms were associated. In the remaining 26 outbreaks the suspected food contained a mixed flora of apparently banal organisms, although the counts were very often high. Forty-six foods from outbreaks were injected intraperitoneally into kittens and twelve caused vomiting. Unheated extracts of 78 strains of bacteria were tested similarly for kitten toxicity in 4 ml. amounts and 45 (58 per cent) caused vomiting with or without diarrhoea. The types of bacteria which proved toxic in kittens were: lactose fermenting and non-lactose fermenting coliform bacilli, *Proteus vulgaris*, *Staphylococcus aureus*, *Staphylococcus albus* and aerobic sporing bacilli. Several strains of micrococci and *Str. faecalis* proved non-toxic. No definite conclusions could be drawn from the observations, owing to doubt of the validity of the kitten test and to the need of elucidation of the mechanism of action of Staphylococcal enterotoxin. The ability of organisms other than *Staphylococcus aureus* to form toxic products in food-stuffs, able to cause food poisoning of the toxic type, is therefore still not proven, though the available epidemiological and laboratory evidence is highly suggestive.

Minett (1938) carried out some important and interesting experiments on staphylococcus food poisoning at the Research Institute in Animal Pathology, Royal Veterinary College, London. In his summary he states:

(1) Feeding tests on monkeys (*Macacus rhesus*), dogs and cats are unsatisfactory for detecting the presence of enterotoxin, owing to the variable susceptibility of these animals by the oral route.

(2) Using Dolman's method, in which the material is injected intraperitoneally into kittens, the production of enterotoxin has been demonstrated by: (*a*) sixteen out of thirty-eight strains of *Staph. aureus*, isolated from cases of acute or chronic mastitis or from normal udder milk; (*b*) four out of five strains of *Bact. coli*, mostly from calves with 'white scours'. No enterotoxin was obtained from fifteen strains of *Str. agalactiæ* from mastitis in cows.

(3) The formation of enterotoxin under natural conditions has been observed: (*a*) In udder milk seeded with *Staph. aureus* or naturally contaminated with that organism and stored at atmospheric temperatures (18° and 22°C.). The substance remains active in cheese prepared from such milk. (*b*) In layer cake made with cream naturally contaminated with *Staph. aureus*.

(4) A small outbreak of poisoning due to potted meat paste was shown to be caused by a non-haemolytic *Staphylococcus*.

(5) A few feeding experiments on man with milk or cream, in which food-poisoning staphylococci had grown, were negative, but on one occasion a *Staphylococcus* from a case of mastitis yielded a culture filtrate which caused symptoms of food poisoning.

(6) Enterotoxin has the following properties. It is resistant to heat (95°C., 30 min.), to low concentrations of formalin sufficient to destroy the haemolytic toxin, to acid (*p*H 5·0), and to rennet, but is destroyed by trypsin. It diffuses freely into the culture medium but only slightly through collodion. It is antigenic. Its properties are such that enterotoxin can be classed as a bacterial exotoxin.

Haynes and Hucker (1946) record the following information on the effects of physical agents on the enterotoxin:

The most prominent physical property of enterotoxin is its heat stability. Eleven years ago there was a brief controversy over this point, Jordan (1930a, b) contending that boiling destroyed enterotoxin and that 60–65°C. for half an hour either destroyed or greatly weakened it and Dack, Cary, Woolpert, and Wiggers (1930) holding that it resisted boiling for half an hour. Jordan, Dack, and Woolpert (1931) soon discovered the error and confirmed the resistance of enterotoxin to boiling. This was confirmed by Dolman, Wilson, and Cockcroft (1936), Minett (1938), and Dack (1939). Rigdon (1938) found that boiling for two hours destroyed the enterotoxin and Davison and Dack (1939), besides confirming the instability of the enterotoxin to prolonged heating, found that autoclaving decreased its potency. Jordan and Burrows (1933) found that it was unstable to heat in acid solutions (N/100 HCl).

Most exotoxins tend to deteriorate when they are stored even when held at low temperatures. Enterotoxin, besides differing from ordinary exotoxin in its resistance to heating, retains its toxicity when stored at low temperatures for two months, as reported by Jordan, Dack and Woolpert (1931) and Jones and Lockhead (1939).

Other physical properties which have been investigated are the ability of enterotoxin to dialyse, its adsorptive capacity, and its volatility. Although it diffuses freely into the culture medium, enterotoxin diffuses only slightly through collodion, according to Minett (1938) and Jordan and Burrows (1933). It is partially adsorbed on Seitz pads during filtration, but to a lesser extent than the other toxic factors present in micrococcus filtrates, as shown by Woolpert and Dack (1933). Lastly it was found by Jordan and Burrows (1933) that enterotoxin would not distil.

Regarding the effect of low temperatures on enterotoxin, Jones and Lockhead (1939) found that out of 50 strains of micrococci isolated from frozen vegetables, 12 produced enterotoxin. Phillips and Procter (1947) observed that staphylococci survived freezing in cream food products. Gunderson and Rose (1948) in a

study found that in some cases exceptionally high numbers of bacteria were found in commercially produced chicken chow mein and chicken salad despite prolonged storage at – 12° to – 14°F. (– 24°–44°C. to – 25°–55°C.). Five out of eight samples of chicken salad examined contained potential food-poisoning haemolytic *Staphylococcus aureus*.

The results of experimental researches of various investigators go to show that frozen foods are liable to contamination by enterotoxin-producing staphylococci which are not destroyed by freezing and that these organisms will grow in suitable food products. If the latter are exposed to higher temperatures for a sufficient length of time and under favourable conditions enterotoxin may be produced.

At the present time there is an urgent need for a method whereby staphylococci which produce enterotoxin can be reliably differentiated. Various cultural methods, such as that of Stone, are unreliable. Feeding to rhesus monkeys is sometimes satisfactory, but these animals are not usually available nor are they very sensitive. Kitten feeding and particularly inoculation tests have been much used but, as indicated above, considerable doubt has been cast on their reliability. Human volunteers at times give a satisfactory test, but difficulties arise, however, because individuals vary in their susceptibility to the enterotoxin, as shown by Jordan (1930), Meyer (1934), Shaughnessy and Grubb (1937), Minett (1938). Some persons are quite unaffected, while others develop an immunity; see Barber (1914), Shaughnessy and Grubb (1937). Moreover, volunteers are not always available, the experiments are decidedly unpleasant, and they are not entirely without risk.

Dolman (1943), in the summary of his paper 'Bacterial Food Poisoning,' gives the following:

The hypothesis is advanced that the 'toxin' type of food poisoning outbreak, so frequently reported as of unknown origin, is in reality usually due to enterogenic staphylococci which may be masked, overgrown or even extinct when the peccant food is examined in the laboratory. This hypothesis was strengthened by an experiment in which 3 persons were made violently ill by consuming a saline extract of 2·7 gm. of wiener sausage which had been inoculated with enterotoxigenic staphylococci, and subsequently with Proteus vulgaris. At the time of consumption the wieners were decomposing, and the Proteus counts were 30,000/50,000 million organisms per gram, outnumbering the staphylococci a hundredfold. At a prior stage, when the wieners were already toxic, no staphylococci could be detected on poured plates. Larger amounts of extract from wieners equally infected

with the same strain of Proteus vulgaris, but not inoculated with staphylococci, were eaten without ill-effect by the same 3 volunteers.

Surgalla, Kadavy, Bergdoll, and Dack (1951) carried out investigations to devise practical methods of producing large quantities of enterotoxin, a necessary step preliminary to purification studies and with the object of gaining more fundamental knowledge of the nature of staphylococci enterotoxin.

Regarding the antigenic properties of staphylococcus enterotoxin, recent research and experiments carried out in Canada by Dolman and others (1944) showed that although human beings do not appear to acquire any resistance to the effects of repeated doses of enterotoxin taken by the mouth, *injection* of a few small doses of suitably prepared enterotoxic filtrates *will* provoke active immunity.

A group of seven volunteers showed on the average resistance to at least five times the initial minimal reacting dose of enterotoxin taken by the mouth, after receiving a series of small subcutaneous injections of a formalinized filtrate prepared from a food-poisoning strain. The reactions on the whole were negligible. Protection against staphylococcal food poisoning therefore seems a feasible procedure. Apart from human beings, cats were shown to acquire active immunity following multiple intravenous injections of enterotoxin. The serum of immunized animals also showed neutralizing power against the enterotoxin.

Possible Sources of Enterotoxigenic Staphylococci

Early observers considered the most likely sources to be of bovine origin (meat and milk). Later, other investigators isolated the organism from the udders of cows. Jordan and Burrows (1934) came to the conclusion that raw milk or cream, and also human contact with food, pointed strongly to bovine or human sources. Minett (1938) showed that the enterotoxin subsisted in cheese made from infected milk, and that milk from cases of acute and chronic mastitis was enterotoxic. Shaughnessy and Grubb (1937) found ice-cream made with raw milk from cows suffering from mastitis to be enterotoxigenic.

In recent years, however, research workers and investigators of staphylococcal food-poisoning outbreaks have come to the conclusion that one of the most important sources of the enterotoxin-producing staphylococci is man. See Jordan (1930), Haynes (1935), McCastline, Thompson, and Isaacs (1937), Roberts, Deadman,

Elliott, and Wilson (1938), Roberts (1939), Chapman, Berens, and Stiles (1941).

Foods as Vehicles in Staphylococcus Food Poisoning

Although certain foods are suitable for the growth of staphylococcus without the production of the enterotoxin, others allow the staphylococcus to grow and produce the enterotoxin. Nevertheless, a considerable variety of foods have been incriminated from time to time in staphylococcus food-poisoning outbreaks. Among the foods are: meat canned or processed; fish canned or processed; milk and milk products; pastries such as custard pies, tarts, cream-filled and chocolate éclairs, ice-cream, cakes, chicken salad, sandwiches, liver sausage. Ordinarily the contaminated food is not affected in appearance, taste or smell, hence the difficulty for the observer to detect any signs of contamination.

Coughlin and Bascom Johnson (1940) referring to gastro-enteritis outbreaks from cream-filled pastry, remark:

(1) Outbreaks of food poisoning due to staphylococcus toxins in cream-filled pastries are not uncommon.

(2) During the 5-year period 1935–9 inclusive, 17 outbreaks of gastro-enteritis, involving 1,246 cases due to eating cream-filled pastries, were investigated in New York State exclusive of New York City. Thirteen of these, resulting in 1,227 known cases, were apparently due to staphylococcus.

(3) Five of the outbreaks, accounting for 60 cases, were traced to pastries from a single bakery.

4) Chocolate éclairs and cream puffs were most commonly involved, rarely cream-filled pies.

In a large number of outbreaks in which the above foods were incriminated, it is recorded that the circumstances favoured the multiplication of the organisms. There is no doubt that during preparation, some of the foods mentioned receive a good deal of handling; moreover, certain of the foods prior to consumption are exposed for some time in kitchens, storerooms, shop windows, etc., and are subjected to warm temperatures, conditions most favourable for the growth of the staphylococci in the food.

In this connection Jensen (1944), writing on the 'Incubation Zone', reports:

When the food-poisoning staphylococci grow for a certain length of time, they begin to elaborate an enterotoxin. They do not cause food poisoning if their cells are ingested alone. Experimentally, and in outbreaks, we have determined how long they must grow, and at what temperatures they grow in a suitable medium before they can form

enough gastro-intestinal irritants to produce their more or less characteristic symptoms in man. The temperature range lies between 60°F. and 115°F. The length of time varies roughly from 4 to 8 hours, depending upon the perishability of the food—i.e. on how well bacteria can grow in it. To avoid hazard from growth of these bacteria, it is well to provide a safety margin by considering the incubation danger zone from 50°F. to 120°F.

As all food products carry inoculations of miscellaneous bacteria, it must be assumed that any unprotected food may become contaminated with a type of bacteria that can form substances toxic to man.

Some observers have found that preserved meats, such as ham and tongue, are suitable media for the growth of the organisms. Kelly and Dack (1936) record an outbreak due to contaminated ham sandwiches which had been kept at room temperature for 18 hours.

Oddy and Clegg (1947) described an explosive outbreak of staphylococcal food poisoning in September, 1946 (among 167 miners employed at several collieries), due to the consumption of pressed pickled beef sandwiches. *Staphylococcus aureus* was isolated from an uncut block of the pressed pickled beef, the sandwiches, the vomit of individual cases and from 8 of the workers in the food-preparing centre. This evidence was reinforced by bacteriophage typing. The butcher responsible for pickling and cooking the meat and who handled it from the raw state to the finished article, was found to be harbouring the above organism in his throat and in a small cut on his right hand. The observers stated that 'it seems most likely that from this second source the contamination of the pressed beef occurred.'

Kelly and Dack (1936) have shown that staphylococci will grow in meat with a 10 per cent sodium chloride content. Other observers have found that the organisms multiply in a salt concentration of from 7 to 10 per cent.

Dack, Woolpert, Noble, and Halliday (1931) noted 'that the staphylococcus from a cake was destroyed by baking even at an internal cake temperature of 75°C. (167°F.) for 12 minutes. The oven temperature was 150°C (302°F.). The organism survived when the oven temperature was lowered to 120°C. (248°F.). They were led to believe that the cake filler was contaminated during, or after its preparation, and that the staphylococci invaded the cake from it.

Segalove, Davison, and Dack (1942) carried out an investigation on the 'Growth of a food-poisoning strain of Staphylococcus experimentally inoculated into canned foods.' They stated:

The canned foods studied in these experiments include corn, peas, asparagus, spinach, string beans, tomato juice, peaches, shrimp and

salmon. These foods were chosen since they represent a cross-section of the different types of food on the market; for example, peas and corn are low-acid products; asparagus, spinach and string beans are semi-acid products; whereas tomato juice and peaches are acid products. Shrimp and salmon are examples of sea food, the chemical composition of which is sufficiently different from fruits and vegetables to warrant study.

[Summary]: Canned foods of low-acid content (peas, corn); semi-acid content (asparagus, spinach, string beans); acid content (tomato juice, peaches); and canned fish (salmon, shrimp) were experimentally inoculated with a food-poisoning strain of Staphylococcus aureus. Growth was found to be best in the low-acid foods, but was not found to be affected by the kind of food. In high-acid foods growth did not occur. In almost all cases growth was better at 22° than at 37°C. The staphylococcus produces acid but no gas in the carbo-hydrates which it ferments; however, in canned peas and corn gas is produced. More gas is produced in peas than in corn.

With regard to milk, Dolman (1939–41) remarks:

Cow's milk is the only significant source of endogenous staphylococci in a foodstuff, all other contamination by these organisms being exogenous, a fact which eliminates certain difficulties inherent in the control of Salmonella food infection.

He emphasizes that pasteurization markedly reduces the food poisoning hazard from endogenous staphylococcal infection of milk, but this may fail as a safeguard if the whole process is inefficiently carried out.

Williams Smith (1957) conducted investigations into the multiplication of *Staphylococcus aureus* in cow's milk. In his summary two reasons are given for the low incidence of staphylococcal poisoning originating from raw milk.

These were the comparatively low temperatures at which milk is normally kept and the frequent presence in it of bacteria other than *Staph. aureus*. These two factors were shown to have a limiting effect on the degree of multiplication of *Staph. aureus* that could take place.

Surgalla and Dack (1945) carried out some research concerning the growth of *Staphylococcus aureus* (*Salm. enteritidis* and Alpha-type streptococcus) experimentally inoculate into canned meat products. The following is their summary and conclusions:

Staphylococcus aureus, Salmonella enteritidis and Alpha-type streptococcus experimentally inoculated into test-tube preparations of canned roast beef, corned beef and potted meat grew luxuriantly and survived for at least 60 days at 22° and 37°C. when loss of moisture was prevented.

Staph. aureus inoculated into cans of specially ground and untreated roast beef at a definite point, either on the top surface or in the centre

of the can, spread rapidly throughout the contents of the can. Within the limits of our methods of determination, spread of Staph. aureus throughout the contents of the can from a definite point of inoculation occurred in all instances. The spread was more rapid in roast beef than in the other meat products tested, a finding undoubtedly due to the greater amount of moisture in the can. Growth into the solid slabs of meat was not as rapid as through the ground products.

No abnormal odours were associated with the meats in any of the experiments. When extensive growth of Staph. aureus occurred, the yellow growth was often observed on the surface throughout the meat, and sometimes gave a 'ropey' consistency to the meat products. These results indicate that, in the event of contamination of the types of canned meat products studied, organisms rapidly become distributed throughout the entire contents of the can. Therefore, discarding only portions of the contents of a contaminated can would afford no protection against the bacteria. Likewise, survival of organisms for at least 60 days at both room and incubator temperatures indicates the possible danger of storage of contaminated products at these temperatures.

Cathcart, Godkin, and Barnett (1947) investigated the growth of *Staphylococcus aureus* in commercial dry-mixed puddings (with and without the addition of milk), using various prepared fillings. They found that puddings containing milk supported the growth of the organism, but that growth was inhibited when water was substituted for milk. Fillings such as vanilla, pumpkin, squash, and sweet-potato pie fillings, cheese cake fillings, and whipped-cream mixes were found to support the growth of *Staphylococcus aureus*, but that fruit fillings such as peach and raspberry contained sufficient acidity to check the growth of this organism.

Hussemann and Tanner (1949) compared strains of staphylococci isolated from foods; 40 of the strains were from foods not known to have caused food poisoning, and 28 strains, for comparison, had been accepted as the causative agent in food-poisoning outbreaks. The strains were subjected to certain biochemical reactions with the results given in the table on p. 149.

The strains were also submitted to the kitten test of Dolman and Wilson. Toxins were made using the Dolman method, except that alpha and beta toxins were not destroyed. Owing, however, to the difficulty in obtaining kittens the observers mentioned that the test was of small value because it was so limited. No conclusion was reached from this work with kittens since one of the kittens injected with uninoculated media was positive.

The observers state that

under the conditions of these experiments, however, none of the tests devised seemed to be wholly successful in separating the enterotoxin-

forming from the non-enterotoxigenic staphylococci. Conditions necessary for the formation of enterotoxin are just beginning to be defined. Until techniques are available which permit toxin identification, results of finding staphylococcal in even a food shown epidemiologically to be incriminated in a food poisoning, must remain in question. It has been demonstrated that staphylococci were harbored in foods which were eaten by large numbers of persons and with no untoward results.

Techniques Employed	40 *strains of staphylococci from foods not known to have produced food poisoning*	28 *strains of staphylococci from foods incriminated in food-poisoning outbreaks*
	per cent.	per cent.
Pigment formation on Loeffler's medium	White 32·5 Cream 37·5 Yellow 22·5 Orange 7·5	White 3·6 Cream 17·8 Yellow 3·6 Orange 75·0
Hemolysis on rabbit blood agar	90·0 positive	96·4 positive
Coagulase (rabbit plasma) .	2·5 „	96·4 „
Acid formation on phenol red mannitol agar . . .	75·0 „	100·0 „
Growth on bromthymol blue agar	90·0 „	96·4 „
Growth on 7·5 per cent NaCl phenol red mannitol agar .	97·5 „	96·4 „
Stone's reaction . . .	80·0 „	75·0 „

Segalove and Dack (1951), experimenting with dehydrated meat, found that the

growth of food poisoning strains of staphylococci or streptococci isolated from food poisoning outbreaks was not evident in samples containing 20 per cent moisture or less. When a moisture content of 30 per cent was reached, growth of the organisms was dependent upon the salt content of the sample. High salt content inhibited growth. The staphylococcus strain used grew in all meat containing 40 per cent or more moisture regardless of salt content.

Ludlam (1952) investigated the occurrence of *Staphylococcus aureus* in slaughter-houses. Swabs were taken from carcasses in a large, well-run slaughter-house and *Staph. aureus* was isolated from 113 out of 175 carcasses. The organism was widespread on aprons, knives, and other implements of the slaughterers. Phage-typing showed that the common types in the meat were also the common types on the implements.

The findings suggested that the possibility of slaughterhouse contamination should be borne in mind when attempting to determine

the source of staphylococci found in cases of food poisoning due to meat, especially made-up meat products.

Symptomatology

In cases of staphylococcus food poisoning the symptoms usually occur suddenly, that is, $\frac{1}{2}$ to 5 hours after ingestion of the incriminated material. The time varies, however, according to the amount of the enterotoxin ingested with the food and the susceptibility of the individual concerned. The short period of time between consumption and effect is characteristic of a toxin type of food poisoning.

The illness usually commences with nausea followed by frequent vomiting which occurs quite suddenly. There is considerable retching with abdominal pain and cramp, followed by persistent diarrhoea. At first the stools are loose but later profuse and watery. In mild cases nausea and vomiting may occur without diarrhoea. In severe cases the vomit and stools may be bloodstained.

Dolman (1943) states:

> It is now accepted that the action of the enterotoxin is not directly upon the gastro-intestinal tract, but rather upon some specific area of the central nervous system.

In the early stages of the illness there can be headache and dizziness and sometimes a rise in temperature with rapid pulse; pallor, numbness of extremities, thirst, cold sweats, dehydration, low blood pressure, prostration, and collapse. In some cases the temperature is subnormal and one case has been recorded where it dropped from 98·4° to 96°F. for some hours (Denison 1936).

In the majority of cases of staphylococcus food poisoning, improvement in the condition commences in from 4 to 5 hours and recovery is rapid. In severe cases the gastro-intestinal symptoms often last some hours or even days and prostration is most marked.

Mortality

The mortality rate for staphylococcus poisoning is low, in fact very few deaths are recorded. In America, however, Weed, Michael, and Harger (1943) mention a fatal staphylococcus intoxication from the ingestion of goat milk. Mention of fatal cases is recorded by the following workers: Finnel (1856), Denys (1894), Blackman (1935), Dorling (1942).

Kodama, Hata, and Sibuya (1940) record a premature birth with death of the infant following an attack due to the consumption of fish-sausage.

Prevention and Control

The prevention and control of staphylococcus food poisoning are fraught with considerable difficulties owing to the abundance and widespread nature of the various staphylococcus strains (but, as pointed out before, only certain of these strains of the organism are responsible for the production of the enterotoxin), and to the fact that the infected or intoxicated food is not altered in appearance, taste, or smell; consequently there are no physical signs to indicate whether or not the food is contaminated. Prevention must therefore greatly depend upon such matters as general hygiene and personal cleanliness in all its aspects, and the protection of food during preparation and storage.

In this connection Getting, Rubenstein, and Foley (1944) are of opinion that 'one of the most effective methods of reducing food-borne diseases is the enforcement of proper personal hygiene practices by all food handlers. Keeping the hands away from the mouth and nose, covering the mouth with a handkerchief while coughing or sneezing, followed by washing the hands, covering foods whenever possible, refrigerating those that are perishable, reducing the interval between cooking and eating, eliminating food handlers with purulent wounds, boils, or infections of the hand, preventing food handlers with sore throats from preparing food—all these will reduce most of the instances whereby staphylococci and streptococci may contaminate food.

These observers also give an interesting 'Summary of Epidemiological and Bacteriological Data, 18 Outbreaks, Food Poisoning': 'Our study demonstrates that staphylococcal food poisoning outbreaks may be traced to specific food handlers as sources of infection. In each instance where it was possible to culture the nose and throat of persons responsible for the preparation of an incriminated product, an apparently identical strain of *Staphylococcus aureus* was recovered from at least one of the food handlers and the infected food see [Table]. Although staphylococci are a universal contaminant of the environment, those strains harboured in the nose and throat of the food handler are invariably associated with outbreaks.

The conditions under which contamination of food by staphylococci may take place are usually: A suitable medium (or food)

Outbreak	*Micro-organism*	*Source*	*Ferment*	
			Trehalose	*Sorbite*
1	*Staph. aureus* . .	Food handler .	+	—
	,, . . .	,, . .	+	—
	,, . . .	,, . .	+	+
	,, . . .	Potato salad .	+	—
	,, . . .	Ice cream .	+	+
2	*Staph. aureus* . .	Food handler .	+	—
	,, . . .	Egg salad .	+	—
3	*Staph. aureus* . .	Food handler .	+	+
	,, . . .	Cream filling .	+	+
4	*Staph. aureus* . .	Food handler .	+	+
	,, . . .	Cream filling .	+	+
5	*Staph. aureus* . .	Food handler .	+	+
	,, . . .	'Scotch' ham .	+	+
6	*Staph. aureus* . .	Food handler .	+	+
	,, . . .	Boiled ham .	+	+
7	*Staph. aureus* . .	Food handler .	+	+
	,, . . .	Corned beef .	+	+
8	*Staph. aureus* . .	Food handler .	+	+
	,, . . .	Salad dressing .	+	+
9	*Staph. aureus* . .	Food handler .	+	+
	,, . . .	Boiled ham .	+	+
10	*Staph. aureus* . .	Food handler .	+	+
	,, . . .	Custard pie .	+	+
11	*Staph. aureus* . .	Noodle soup† .	+	+
12	*Staph. aureus* . .	Boiled ham sand.† .	+	+
13	*Staph. aureus* . .	Roast turkey†	+	+
14	*Staph. aureus* . .	Boiled ham† .	+	+
15	*Beta haemolytic strep., group A . . . Griffith, type 2 . .	Food handler .	+	—
		Boiled ham .	+	—
16	*Alpha strep. . .	Food handler .	—	+
	Lancefield, group B .	Cream chicken	—	+
17	*Alpha strep. . .	Food handler .	+	+
	Lancefield, group H .	Baked beans .	+	+
18	*Alpha strep. . . Lancefield, group B .	Chop suey .	—	+

*Present in pure culture.
†Food handlers not cultured.
¹Difference of 1 tube in titration.

ation of				*Alpha Hemo-toxin Titre*	*No. Exposed*	*No. Ill*	*Attack Rate*
Lactose	*Mannite*	*Salicin*	*Entero-toxin*				
+	+	+	+	0·008	200	58	29·0
+	+	+	+	0·008			
+	+	+	+	0·008			
+	+	+	+	0·008			
+	+	+	+	0·008			
+	+	+	+	0·031[1]	211	139	66·0
+	+	+	+	0·016[1]			
+	±	+	+	0·125	Unknown	22+	Unknown
+	±	+	+	0·125			
+	±	+	+	0·125[2]	Unknown	9+	Unknown
+	±	+	+	0·125[2]			
+	+	+	+	0·125	2	2	100·0
+	+	+	+	0·125			
+	+	+	+	0·004	800	180	22·2
+	+	+	+	0·004			
+	±	+	+	0·008	Unknown	30	Unknown
+	±	+	+	0·008			
+	—	+	+	0·031	52	48	92·3
+	—	+	+	0·031			
+	+	+	+	0·008	40	28	70·0
+	+	+	+	0·008			
+	+	+	+	0·031[1]	Unknown	60	Unknown
+	+	+	+	0·063[1]			
+	+	+	+	0·016	2	2	100·0
+	—	+	+	0·008	70	17	24·3
+	+	+	+	0·031	17	17	100·0
+	+	+	+	0·008	3	3	100·0
+	+	+	—[3]	. . .	182	102	56·0
+	+	+	—[3]	. . .			
—	—	—	—[3]	. . .	Unknown	18	Unknown
—	—	—	—[3]	. . .			
+	+	+	—[3]	. . .	6	6	100·0
+	+	+	—[3]	. . .			
+	—	+	—[3]		3	3	100·0

[2]Weak enterotoxin in casein hydrolysate media. (Checked through kindness of Dr. G. M. Dack, University of Chicago.) Toxicity enhanced by culture in cream filling.

[3]Whole cultures and tissue media filtrates enterotoxic for kittens.

in which the specific strain of the organism will grow well and in sufficient numbers, and the exposure of the contaminated medium to a suitable temperature for a sufficient length of time to produce the enterotoxin.

Jensen (1944), writing on the 'Care of Foods,' makes a number of useful recommendations, as follows:

As a further safeguard against food-poisoning hazards, the foods should be cooked carefully according to instructions, so that these bacteria from chance inoculation are destroyed. As an example, hams should be cooked so that the inside reaches a temperature of at least 162°F. We must apply these temperatures to the meat just like [as] the dairy operator must pasteurise milk and cream to safeguard them. In this connection, we have seen mess halls and galleys where bone-in, smoked hams, for instance, were held at a temperature of 36° to 40°F. in the meat coolers, and, while there, sawed raw (beginning at stifle joint) on circular and band saws into slices, the slices placed in pans in the cooler, then taken to the steam pressure cookers and cooked at 8 to 10 lb. pressure for about 30 minutes. The ham slices were then served and were never held in the incubation zone for over 3 hours. . . . Contamination of adequately cooked hams occurs during the slicing operations and after, and when the slices are held in the incubation zone for over 4 hours, enterotoxin can form if poisonous staphylococci were present.

When sandwiches are prepared, never allow the bread or filler to remain at danger temperatures (50° to 130°F.) for a longer period than 4 hours. The dangerous bacteria (staphylococci) grow well in most fillers, and especially well in moistened bread. It has been shown in some large outbreaks that the bread in the incubated sandwiches become poisonous as well as the sandwich filler. Never keep the slices of bread moist with a damp cloth as is so frequently done. If the filler is ham or cured or smoked tongue, fried egg, egg salad, fish, sausage, etc., special care must be exercised so that the filler is freshly prepared from materials that have not been in the incubation zone longer than a few minutes.

Soup stocks. Hold-over soups are frequent offenders. Experiments have shown that if soups must be held over, the liquid must be cooled rapidly to 50°F. and then held in a 36°–38°F. cooler not longer than 3 days before use. At 42°F., these stocks can spoil in less than 48 hours.

The following situations should receive special attention: exposure for any length of time without adequate protection in shops, restaurants, canteens, bakeries, kitchens, cook-houses, etc., of those kinds of food especially prone to contamination by staphylococci, at warm or even room temperature.

In canteens, refreshment clubs, etc., much food is prepared and cooked the day before it is eaten and re-heated on the following day before being served for consumption. During this interval, the enterotoxin may develop; it is not destroyed by heat, except at a temperature above boiling point. The difficulty of implicating

the infected food, when the staphylococci have been destroyed, can be appreciated.

Strict cleanliness and the minimum amount of handling in the preparation of all foods should be observed. The avoidance of undue exposure (without protection) of the finished article during the cooling process, is essential. All persons who handle food should scrub their hands with soap and hot water before commencing work and especially after visits to the lavatory.

Concerning the importance of the proper handling of food to prevent contamination, Hussemann and Tanner (1947) examined 380 samples of ingredient and prepared foods of all descriptions, for the presence of staphylococcus. These samples were collected indiscriminately from private homes, cafeterias, lunch counters, restaurants, commercial bakeries, and other service units. The results are as follows. Of the 160 samples of ingredients (such as milk, fresh and canned; cocoa; eggs, dried, frozen, and fresh; cornstarch and flour; pie mixes; sugar), 19, or 10·8 per cent were positive. Of the 220 samples of prepared foods (which included meat and poultry, cream-filled desserts, sandwich fillings, gravy, salad dressing and sauces) 44, or 20 per cent yielded one or more strains of staphylococci. The investigators remark:

> The incidence rate as determined here is undoubtedly only minimum. It is not known with certainty whether or not all *staphylococci* are potential enterotoxin formers, given the proper environment. It is more likely that only certain strains of *staphylococci* produce enterotoxin, but as yet there is not a way of segregating these strains. With so important facts unknown, the implication of the knowledge that at least 20 per cent of foods served in public places were infected with *staphylococci* is obvious. These foods, apparently properly handled, did not as far as was known cause illness. Mishandled, they do form potential foci of intoxication, which could send staphylococcal food poisoning incidence to an even higher level.

Other precautions include the following:

Use of a refrigerator in which to store foods temporarily.

Exclusion from employment of persons in kitchens, food preparation and serving rooms, bakeries, dairies, etc., who may be suffering from affections of the throat (habitual sneezing and coughing), discharges from the nose or ears, boils, surface injuries to the arms or hands, or gastro-intestinal affections.

Efficient pasteurization of all milk and cream.

Prevention and destruction of flies, cockroaches, rats, and mice. The use of poisonous baits in culinary departments, for the eradication of vermine, however, is not recommended.

Pasteurization of milk is of course a necessary and sufficient safeguard against infection from staphylococci, but as pointed out by Dolman (1939–41):

> Prompt, maintained cooling is an important supplementary precaution. Pasteurisation alone may fail as a safeguard, owing to staphylococci having survived an inefficiently carried out process; or through the heat-stable enterotoxin having been elaborated during improper storage of the milk prior to pasteurisation, as in the outbreak recently recorded by Caudill and Meyer (1943). Moreover, pasteurisation will not prevent outbreaks due to subsequent exogenous contamination. Staphylococci introduced exogenously into milk or a milk product can be held from elaborating sufficient amounts of enterotoxin by refrigeration of the foodstuffs immediately after its preparation, as well as of the ingredients prior to their admixture.

Jones, King, Fennel, and Stone (1957), in experiments to study the rate of growth of *Staph. aureus* in cow's milk, found that, in milk from the udder of a cow naturally infected with staphylococci, and in milk drawn aseptically from the udder of a normal cow, staphylococci grew well at 30° and 37°C., but that below 25°C. growth was so slow as to render the chances of the milk becoming toxic within 24 hours practically negligible. They concluded

> that staphylococcal food poisoning is unlikely to occur unless milk from a naturally infected udder is withdrawn under food hygienic conditions; is kept at a fairly high temperature for a sufficient length of time, is not mixed with that of other cows to a degree sufficient to dilute the toxin below the actual level, and is drunk raw. Since these conditions seldom occur in practice in this country, staphylococcal food poisoning caused directly by milk is necessarily uncommon.

The effect of cooking on the survival of staphylococci has been investigated by various observers. Dack, Woolpert, Noble, and Halliday (1931) found that staphylococci introduced into either sponge or butter cake were killed during the baking process. All viable cells seemed to be destroyed when the interior temperature of the sponge cake batter reached 75·2°C. Staphylococci introduced into cream filling were said by these authors to be destroyed during cooking.

Coughlin and Johnson (1941) suggested that in order to prepare custard mix (cream filling) safely, it should be heated to not less than 200°F. for 10 minutes and then cooled promptly.

Cathcart, Merz, and Ryberg (1942) reported that cell suspensions of *Staphylococcus aureus* inoculated into the 'thickening mix' of custard (cream filling) were destroyed by bringing the product to the boil.

It was suggested by Stritar, Dack, and Jungewaelter (1936) and Korff (1936), that cream-filled pastries could be re-heated to destroy staphylococci without damaging the pastries. The former observers investigated the heat penetration into such pastries and recommended the use of ovens heated to 190·6°C. to 218·3°C. for 30 minutes. Cells of staphylococci inoculated into the cream filling were found to be destroyed by such treatment. Maximum temperatures for the cream filling within the pastry of 75°C. for 5 minutes and 76·7°C. for 6 to 8 minutes were recorded for uncovered and covered pans respectively. Gilcreas and Coleman (1941) using inoculated custard (cream filling) observed that no viable staphylococci cells were demonstrable following the rebaking of éclairs. They recommended reheating the custard filling in éclair shells for 15 minutes at a temperature from 216° to 222°C. (420° to 428°F.).

Hussemann and Tanner (1947) made further investigations in order to try and establish the thermal death time temperature relationships for staphylococci in cream filling and to correlate such findings with the range and duration of temperatures attained in accepted cooking procedures for cream filling. In their summary and conclusion they state:

In the range of temperatures studied, *Staphylococcus aureus* strain C12069 (Dolman) was found apparently to survive for 30 minutes at 55°C. and to be destroyed in less than 3 minutes of heating at 85°C. In phosphate buffer solution, PH_7, the organism survived somewhat less stringent heat treatment. It appeared that at the conclusion of such a cooking procedure cream filling was free from viable staphylococcus.

See also Cathcart, Godkin, and Barnett (1947); Abrahamson, Field, Buchbinder, and Catelli (1952).

Illustrative Outbreaks

Cooper (1943) tells of staphylococcal food poisoning from meat pies at Dorchester:

On 5th October, 1943, at about 10 a.m., one of the members of a cottage household, which comprised Sidney aged 60 years, Henry aged 24, Roy aged 19, and Mabel aged 16, purchased three meat pies in a local country town. The pies consisted of minced beef enclosed in pastry. At 1 p.m. the same day all four members of the household had a meal which included the meat pies. Five hours later Roy was taken ill with vomiting and diarrhoea, which passed off leaving him well enough to work the following morning, 6th October. On this same day (6th October) Mabel and Roy had more meat pie at home for their midday meal, Sidney had a portion at his work in the fields, and Henry took some for his dinner at a local factory. At 2.30 p.m. Sidney became

ill with diarrhoea and vomiting, went home and was seen by his own doctor who, suspecting food poisoning, sent his vomit to the laboratory for examination. At 3 p.m. Henry became ill and sought his own doctor at the surgery but failed to find him. He was obviously so ill that he was sent to the Dorset County Hospital and admitted. There he was regarded as suffering from food poisoning, and the remains of the suspected pie, together with the man's faeces, were sent for examination. Mabel, although having eaten pie on two occasions, suffered no ill effects. Sidney and Henry both made rapid recoveries. From Sidney's vomit the only significant findings was the presence of Staphylococcus aureus. In none of the cases did examination of the faeces reveal any members of the typhoid, Salmonella, or dysentery groups, nor were staphylococci found. As a precaution, about a week later, blood was taken from Henry and full agglutination tests were performed, with negative results. On superficial examination, the pie from which only Henry had eaten did not appear abnormal, but a plate count yielded 870,000,000 colonies per gram, of which 480,000,000 were Staphylococcus aureus. Subcultures of staphylococci from the pie and from the vomit (Sidney) were sent to Oxford for phage identification and were reported by Professor G. S. Wilson as belonging to the same type, provisionally referred to as 4/6/47.

Inquiries as to the source of the meat pies revealed that they were manufactured in a neighbouring town 24 hours before they were sold by the retailer. The personnel employed in filling the pies consisted of four persons, all of whom were apparently normal, except that one was reported on examination to have an unhealthy looking nasopharynx. Nose and throat swabs were taken from these four persons, and from two of the nose and one of the throat swabs coagulase-positive staphylococci were isolated. One of the nasal carriers was the person already reported as having an unhealthy nasopharynx. Subcultures were sent to Professor Wilson for phage testing. One of them—from the unhealthy nose—was found to belong to the same phage type 4/6/47 as that to which the meat pie and vomit strains belonged; the others were different.

The most notable feature of this outbreak is the evident value of phage typing of staphylococci in tracing individual strains. If, on further trial, this method is found to be reliable, the statement by Savage (1942) in a recent publication that in staphylococcal food poisoning, 'examination of materials from the patients is not helpful,' will require some qualification. A second point of interest is the much more rapid onset of symptoms in the case of Sidney than in that of Roy, presumably as the result of 24 hours' further 'incubation' of the pies. It is known that this batch of pies numbered at least six dozen. No other complaints have arisen, though the exact distribution of the pies could not be traced. The absence of symptoms in Mabel, who ate pie twice, needs explaining, as does also the fact that Roy was ill after the first pie meal, yet not after the second. Possibly only two of the the three pies were toxic.

The following outbreaks of staphylococcal food poisoning (*Staphylococcal aureus*) are referred to by Savage (1943):

The first outbreak was at Abingdon in May, 1943, and included six cases of food poisoning from one batch of brawn and one case from a

second batch, sold seven days later. The incubation period was 3–4 hours and the symptoms were acute, with vomiting and diarrhoea. Both samples of brawn contained very large numbers of coagulase-positive Staphylococcus aureus. An employee who prepared the first batch of brawn had had a sore throat when preparing it, and a throat swab showed moderate numbers of coagulase-positive Staphylococcus aureus, and this organism was also isolated from a pimple on his wrist. The conditions under which the brawn was made offered extensive opportunities for handling by this man, and the brawn, after being put into tins, was not subsequently treated by heat. From the third batch of brawn made by him under observation on 17th May, coagulase-positive Staphylococcus aureus was isolated.

The other outbreak at Barnstaple, in June, 1943, was also from brawn and involved 10 persons with the usual symptoms, and with a $2\frac{1}{2}$–4 hours' incubation period. The brawn was examined in the laboratory the same day as the outbreak and contained 1,500 million staphylococci per gramme (75 per cent Staphylococcus aureus). Nasal swabs were taken from the three persons who prepared the brawn and one showed a profuse growth of Staphylococcus aureus in pure culture. This strain and the one from the broth were both coagulase-positive. Serological and bacteriophage methods show they were both of the same type and were also the same as the strain from Abingdon.

The brawn was made in the usual way and after boiling was turned into moulds, in which it lay at shop temperature until it was sold next day. It is suggested that the chronic nasal carrier contaminated the brawn during moulding and that the organisms then multiplied rapidly.

Duncan (1944) records an outbreak of food poisoning caused by staphylococcus enterotoxin, which was due apparently to sporadic contamination of individual slices of ham by a nasal carrier of staphylococci, who was responsible for carving the ham after cooking:

In the autumn of 1943 an outbreak of acute enteritis occurred among the personnel of a Civil Defence Column housed in a country mansion, in which 40 out of 200 persons were affected. The symptoms, which developed between 2 and 4 hours after eating a breakfast consisting of porridge, cold sliced ham, bread and margarine with tea, were vomiting and diarrhoea—the latter commencing about $\frac{1}{2}$ to 2 hours after the vomiting—with, in many cases, generalised abdominal pain. All the affected persons were immediately removed to Park Prewett Hospital, Basingstoke, under the care of Dr. J. Simon, who states that, on admission of the patients to hospital, the symptoms varied between slight vomiting, with or without diarrhoea, and very severe retching and vomiting with copious watery bowel evacuations. In some cases the vomited matter and stools contained visible blood and mucus. All the cases were treated as in-patients and, according to the severity of symptoms, were given castor oil, castor oil and opium, opium alone, or morphine and intravenous glucose saline. Three patients were sufficiently collapsed (systolic pressure about 80 mm., and pulse rate between 50

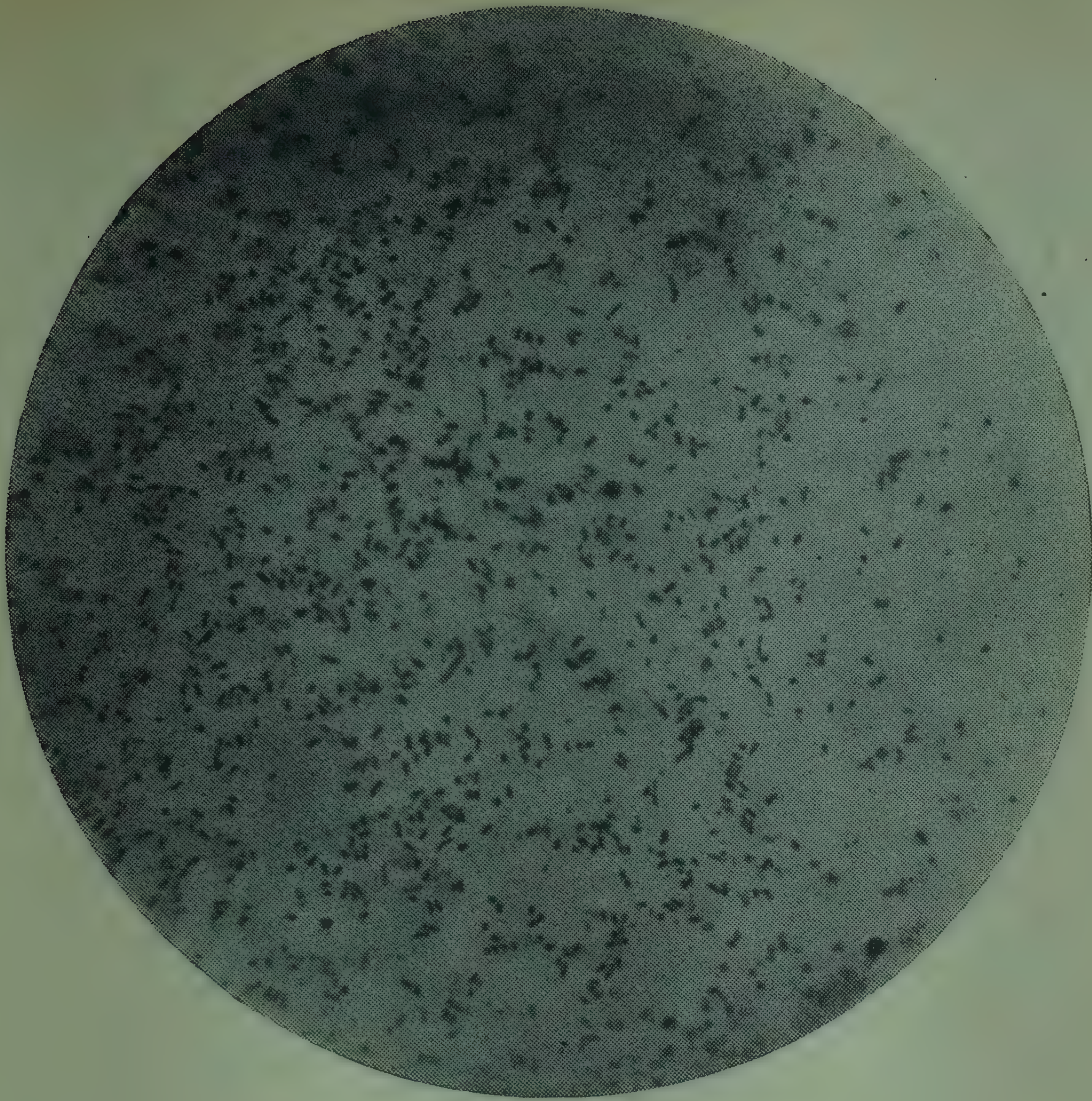
and 60 per minute) to require intravenous glucose saline drip. A small proportion of the patients had temperatures of 100° to 101°F. in the evening, the temperature being still above the normal limit on the day following. A characteristic feature of the more severe cases was low blood pressure and bradycardia persisting for 3 or 4 days after cessation of other symptoms. The majority of the patients were fit for discharge after 3 or 4 days, but a few were kept in for about a week.

Bacteriological and other examinations.—Circumstantial evidence pointed to the breakfast and especially to the cold ham as the probable source of 'infection,' despite the fact that nearly all of the 200 members had eaten of the ham. Samples of all foods consumed within 24 hours of the onset of symptoms were obtained and examined, and also three specimens of vomited matter and five of faeces. No food-poisoning bacterium was found in any of the samples, nor *Clostridium* in any of the foods, but the cold ham and one specimen of vomited matter yielded a haemolytic and coagulase-positive strain of *Staphylococcus aureus*. A plate count made on the ham resulted in the development of approximately 20,000,000 colonies of this organism per gram of meat. Chemical analysis failed to reveal any inorganic poison.

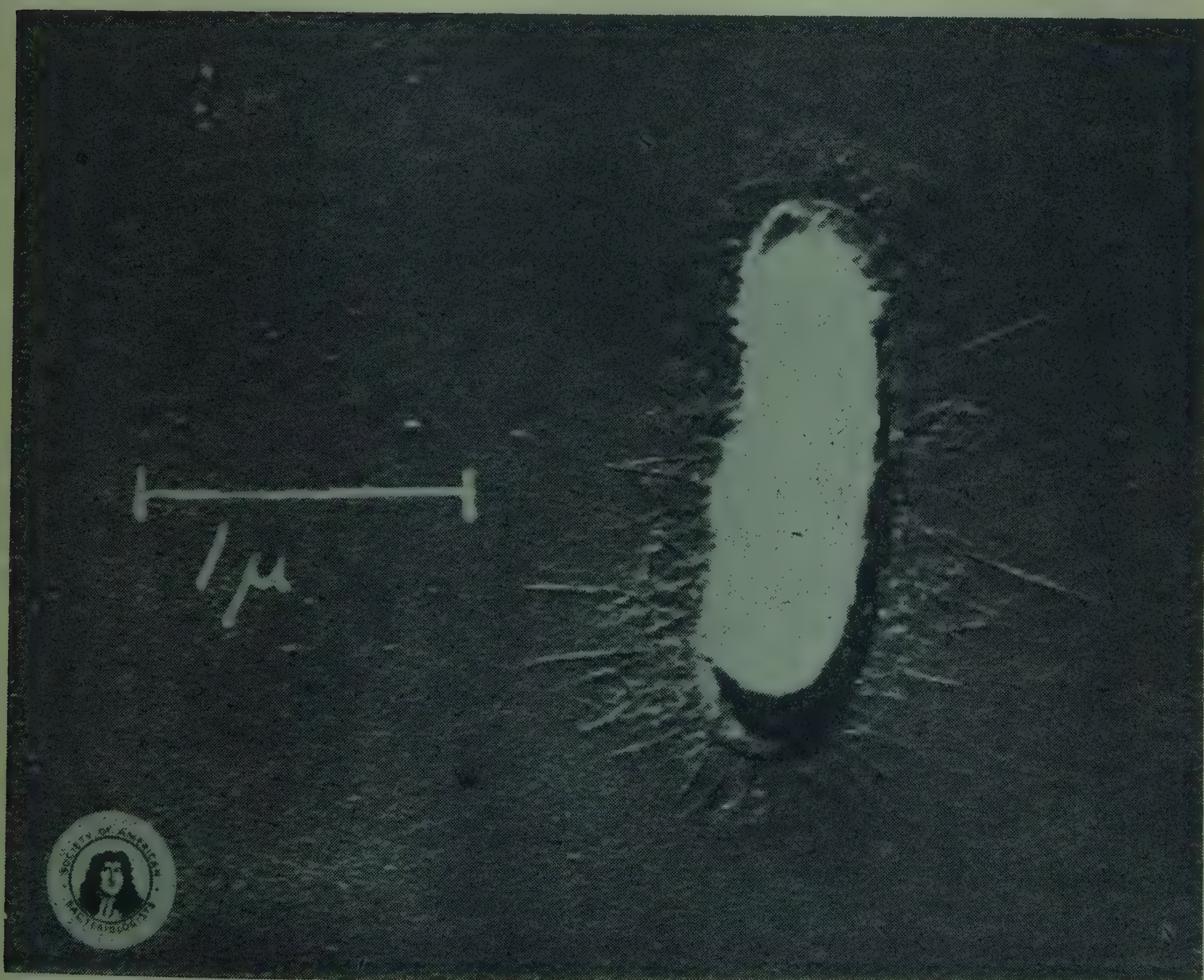
Throat and nose swabbing of the eight members of the kitchen staff yielded rich growths of haemolytic and coagulase-positive *Staphylococcus aureus* from the nasal swabs of four. However, only the culture from the nasal swab of 'X' was found to correspond in biochemical and specific serum agglutination reactions (Serological Type C III) and phage typing (Provisional phage Type 4/47) with the cultures from the ham and vomited matter. X had carved the 200 portions of cooked ham on the day preceding that of the food poisoning. The sliced ham, presumably contaminated by nasal spraying or by fingers contaminated with nasal matter, was kept in warm surroundings overnight, allowing active growth of the coccus and elaboration of its toxin. No doubt, unequal distribution of the toxin in the portions of ham accounted for the escape of 80 per cent of the 200 persons who ate it.

Allison, Hobbs, and Martin (1949) recorded a widespread outbreak of food poisoning due to contamination during manufacture of glazed liver sausage (cooked sausage loaf coated with gelatin glaze) with enterotoxin-producing staphylococci. Some 441 cases were reported from the eastern half of the country. Staphylococci of the same phage and serological types were isolated from samples of vomit and stools of victims of the outbreak and from 102 samples of the sausage meat recovered from shops and homes in different parts of the country.

The staphylococci isolated from the sausage were present in large numbers in the gelatin glaze and absent in the meat. Staphylococci of the same phage and serological type as were isolated from victims of the outbreak and from samples of the infected sausage were also isolated from hands or nose and hands of several operatives who had frequent manual contact with the

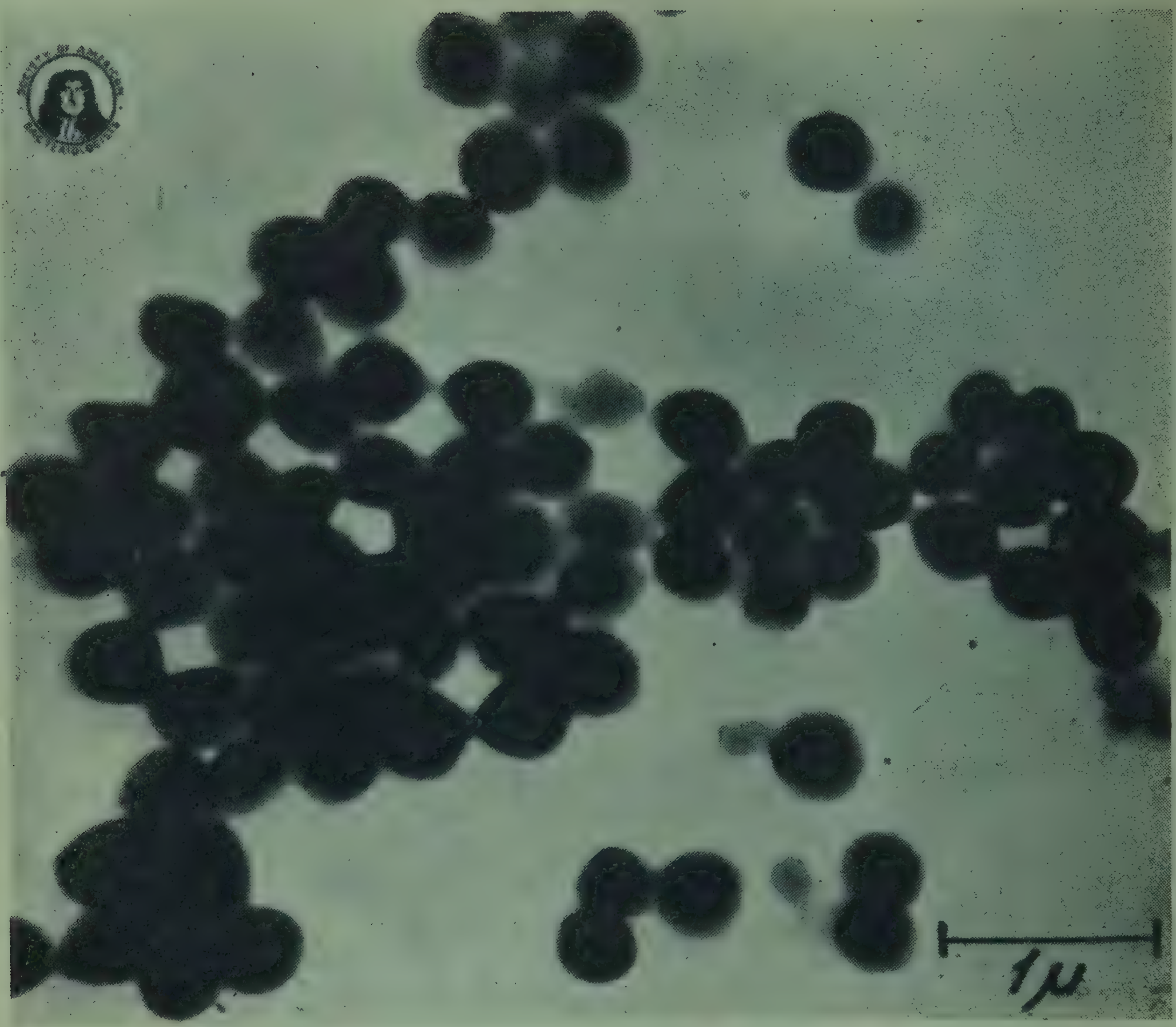


(*a*) *Salmonella typhi-murium*

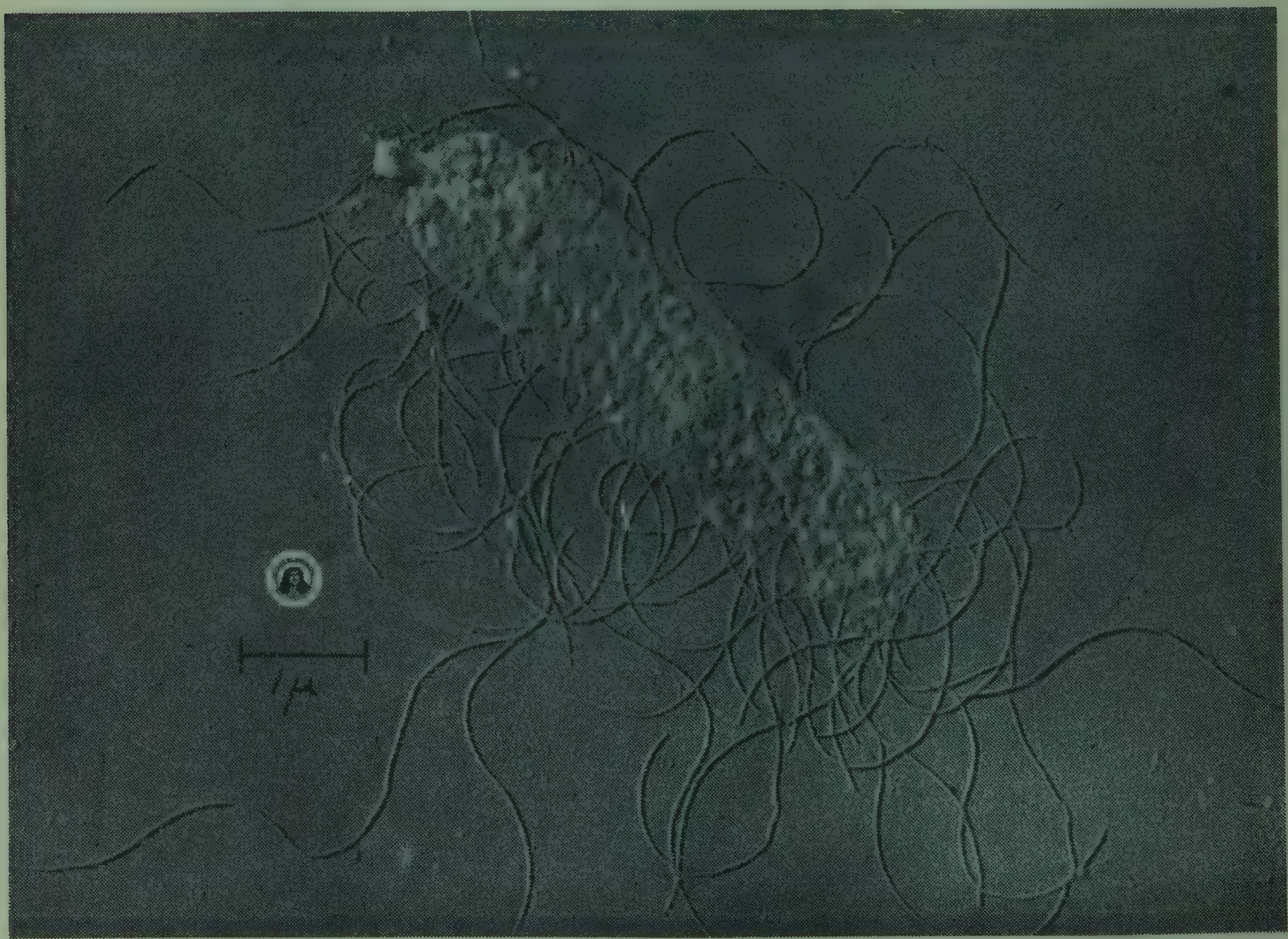


(*b*) Electron micrograph, *Salm. typhi-murium*

PLATE 9

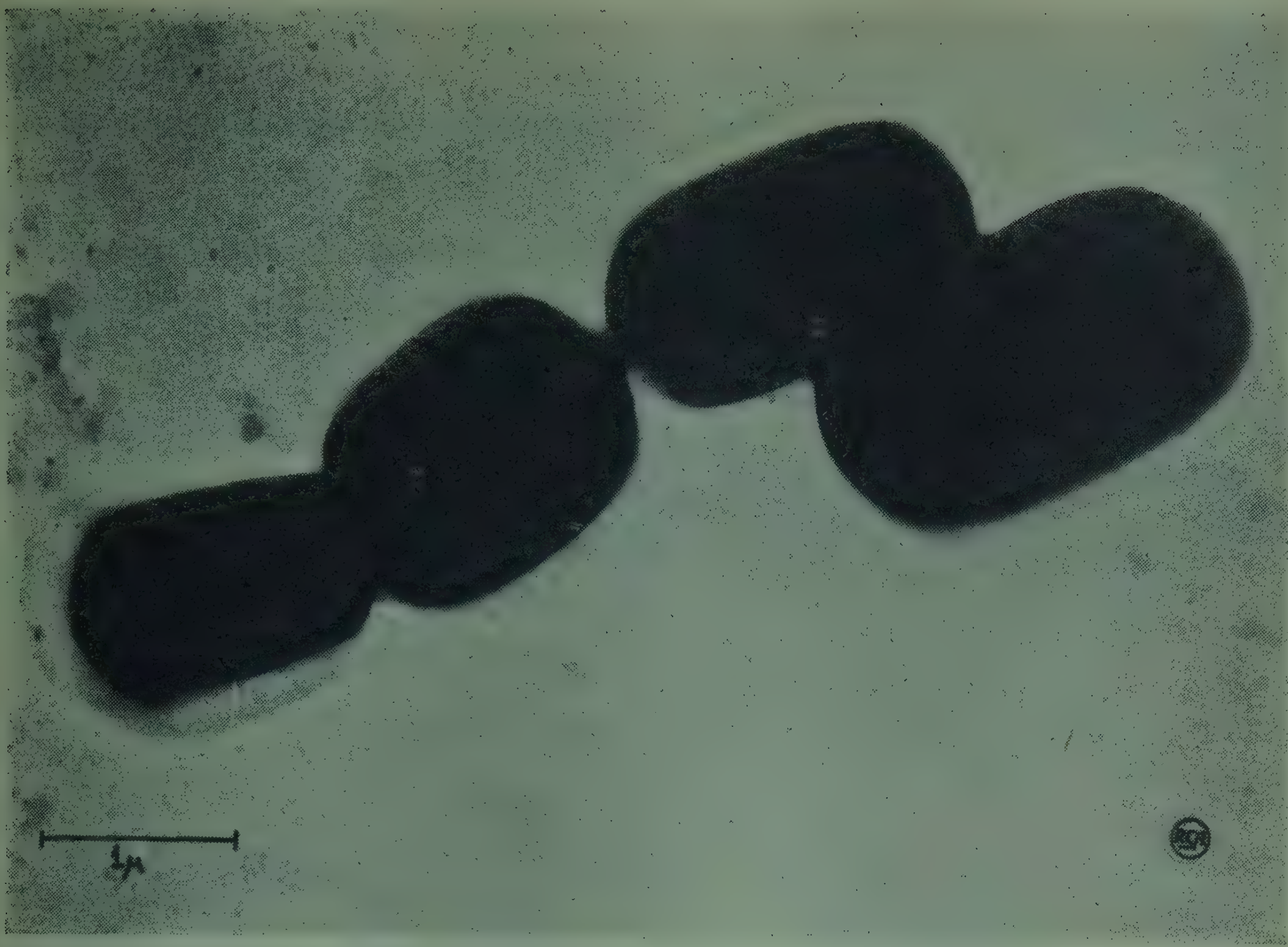


(*a*) Electron micrograph, *Staphylococcus aureus*



(*b*) Electron micrograph, *Proteus vulgaris*

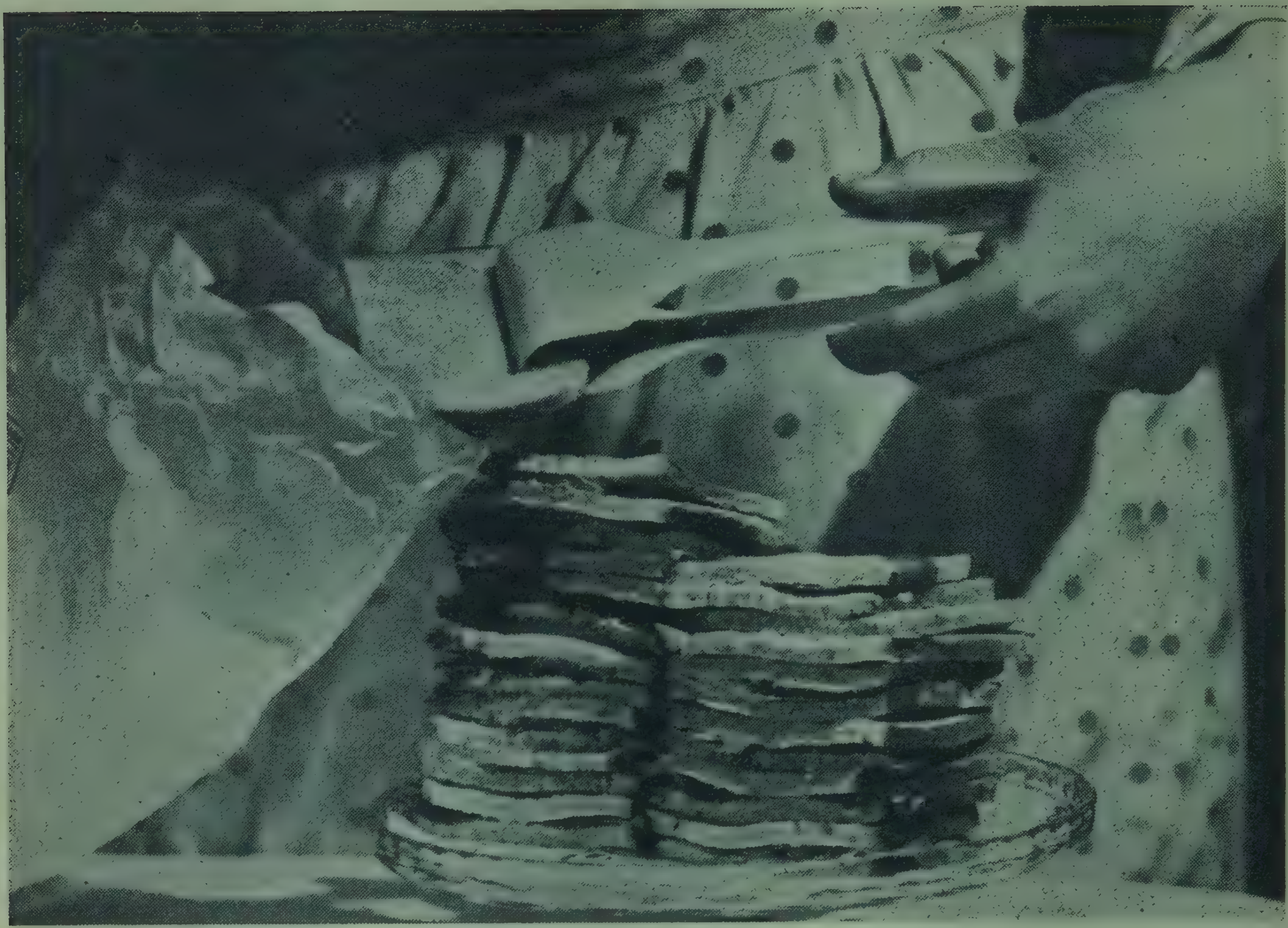
PLATE 10



(*a*) Electron micrograph, *Clostridium perfringens*

(*b*) A violent unstifled sneeze

PLATE 11

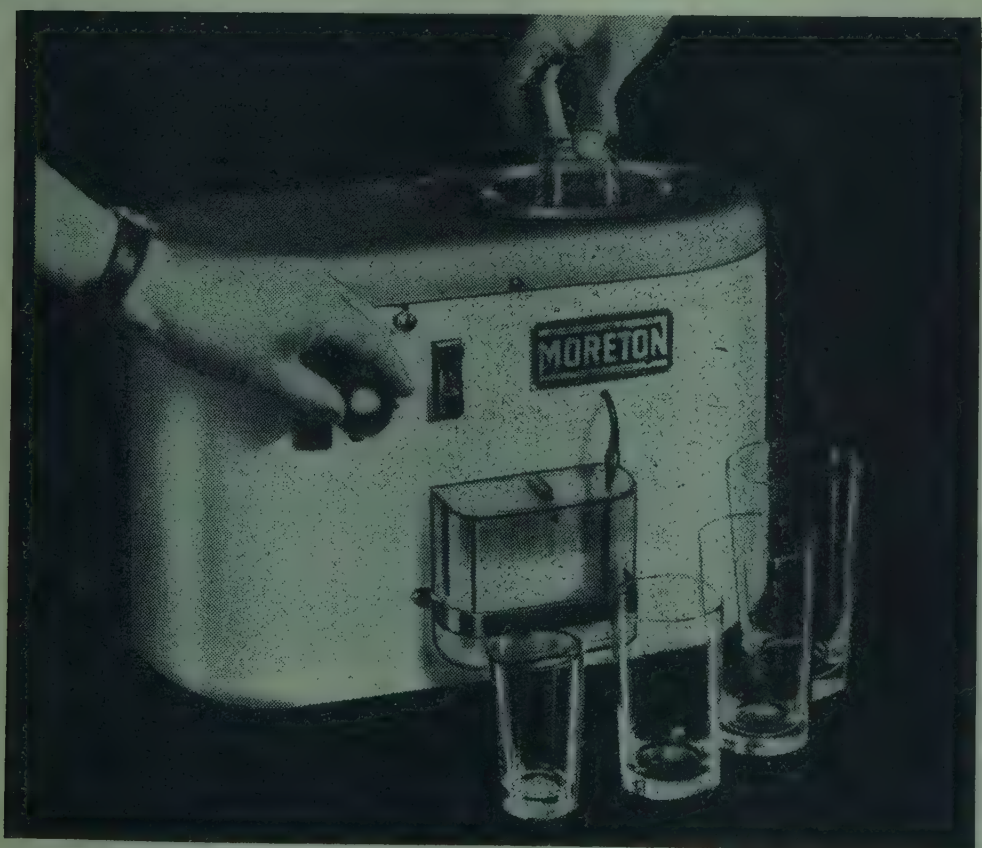


(*a*) Tongs for serving or transferring food

(*b*) Protection for non-infected cut finger

PLATE 12

(a) Bread-wrapping machine



(b) Glass-washing and polishing machine

PLATE 13

(*a*) Steel frame welded to chassis

(*b*) Meat hung in position in container

(*c*) External view

PLATE 14. Modern transport for meat carcasses

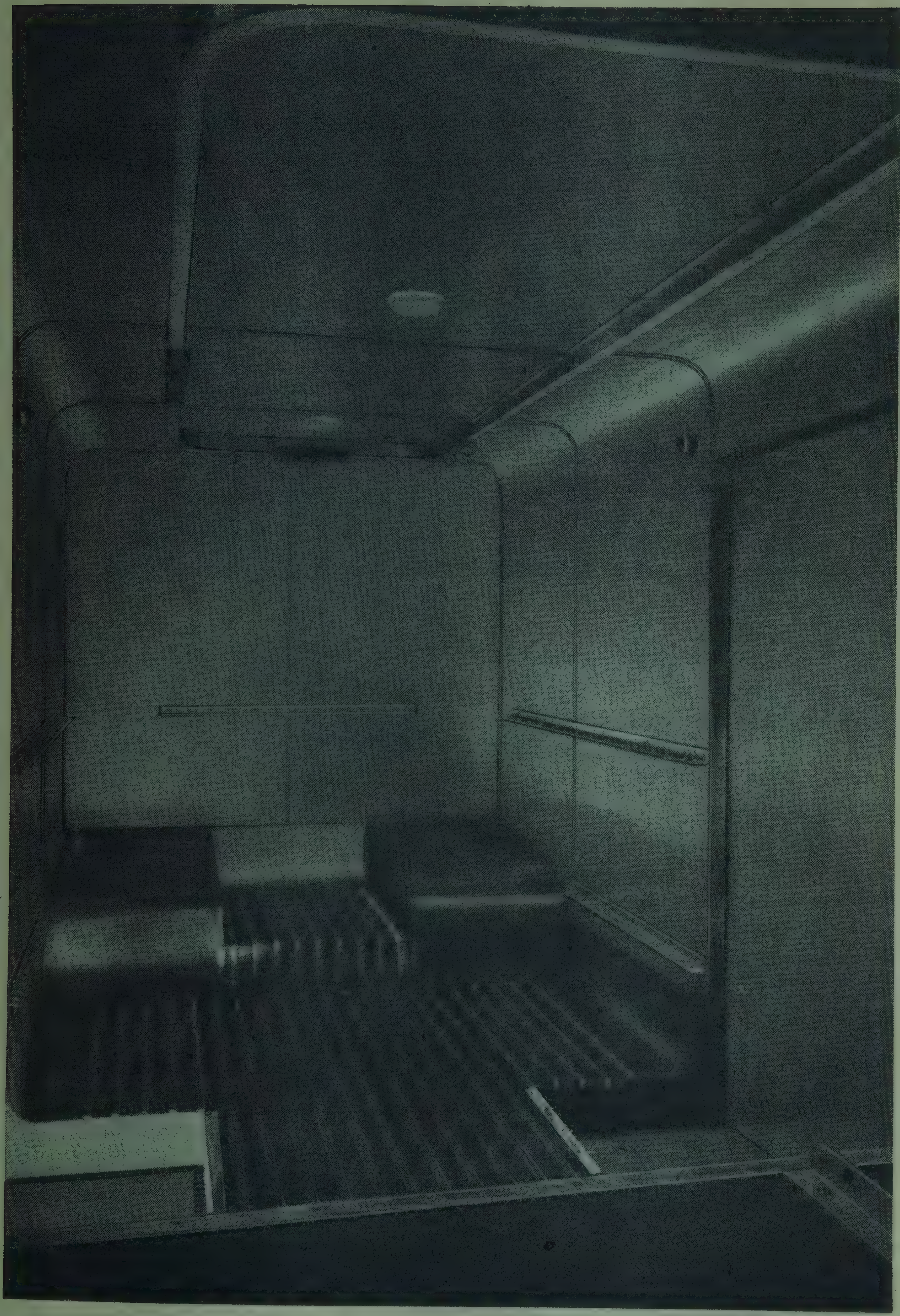
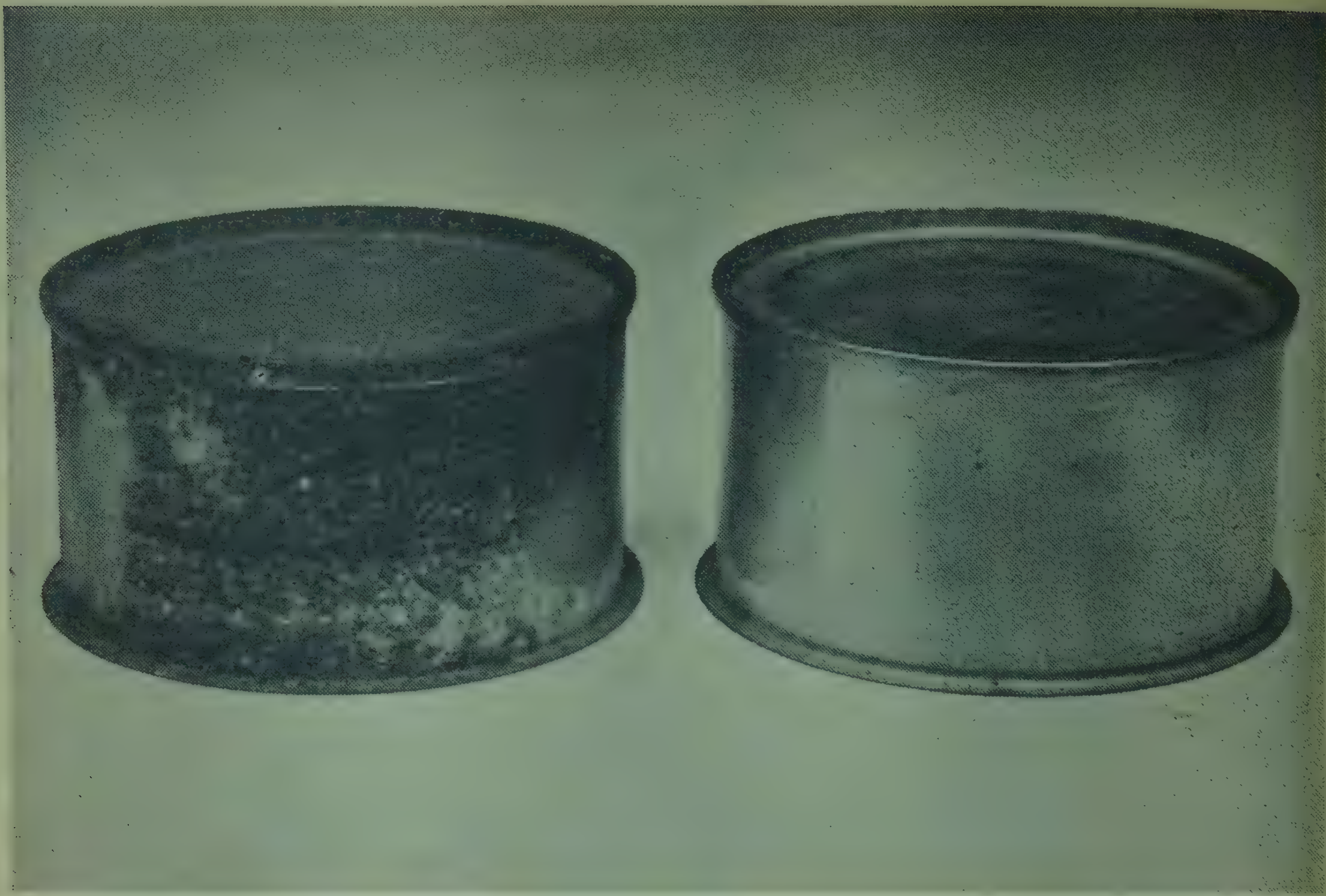


PLATE 15. Modern transport for meat, interior view



(*a*) Untreated can (left) and can treated by the 'Protecta tin' process (right) after exposure to twenty-seven days of wet weather

(*b*) These cans contained fresh peas: the stained cans being of plain tin-plate, the bright one having a protective film

PLATE 16

glaze. The infection of the glaze was attributed to one worker who manufactured the glaze and used his hands to mix the ingredients; this worker harboured the infecting strain of staphylococcus in his nose and on his hands. The low temperature to which the melted glaze was allowed to fall and the high atmospheric temperature prevailing at the time were considered to be factors contributing to the heavy infection of the food.

Macdonald (1944) records the following outbreak of staphylococcal food poisoning caused by cheese made from goat's milk:

On 16th April, 1944, five people had tea, at a little after 6 p.m. in a house in Norwich. Four of the five people lived in the house and had all their meals there; the fifth, Mrs. Br., was a visitor, and had only the one meal in the house. At 10 p.m. on the same evening Mrs. B., wife of the house-holder, became suddenly ill with violent abdominal pain and continuous uncontrollable vomiting. She complained of dizziness and severe diarrhoea, and these symptoms continued throughout the night and to a lesser degree on the following day. At 10.10 p.m. on the same evening her husband became ill with the same symptoms, but he also complained of shooting pains in the legs and abdomen. Mrs. F., another member of the household, became ill at 11 p.m. with similar symptoms, but Mr. F. was not affected. Mrs. Br., the visitor, returned home after tea, and at 10 p.m. she also became ill. All of the patients complained of the extreme severity of the attack and of the acute anxiety which it caused, but all had completely recovered two days later.

The story of this small outbreak suggested food poisoning of the toxic type, and investigation of the patients and of the foodstuffs consumed on the 16th was undertaken. Throat, nose and hand swabs and specimens of faeces were obtained from all members of the household and from the visitor. Unfortunately no specimens of vomit had been kept for examination. Organisms of the Salmonella, dysentery and enteric groups were not found in any of the samples of faeces, but *Staphylococcus aureus* was isolated from the faeces of Mrs. B. Of the possible source of infection, cheese, made by a friend from goats' milk, seemed the most likely and the remainder of the cheese was examined for pathogenic organisms. This cheese had been kept in a cool household larder in the 24-hours' interval before it was sent to the laboratory. Four samples in the cheese, two from the external surface and two from the centre, gave counts of 18, 24, 36 and 24 million *Staphylococcus aureus* per gram. Mr. F., the only member of the household who escaped illness, had not eaten any cheese.

Investigation of the conditions under which the cheese was made showed that the hands of the milkers were carefully washed, and that the teats of the goats were cleansed before milking. Throat, nose and hand swabs taken from those concerned in milking or cheese-making failed to reveal *Staphylococcus aureus*, and no lesions could be found either on the hands of the milkers or on the udder or teats of the goats. From the freshly-drawn milk of one animal, however, 200 *Staphylococcus aureus* per ml. were isolated. The method of cheese-making was to add

rennet to fresh milk and then to strain the whey overnight through a clean muslin bag. The cheese was then pressed into shape. It was stated that the milk from which the suspected cheese had been made was freshly drawn, but experiments suggested that the multiplication of *Staphylococcus aureus* was much more likely to have taken place in the milk itself than in the cheese.

The three strains of *Staphylococcus aureus*—from the cheese, goats' milk and faeces of Mrs. B.—all gave the same biochemical reactions and were all coagulase-positive. Culture filtrates of the cheese strain produced severe abdominal pain and diarrhoea in a human volunteer, whereas a similar amount of filtrate after boiling had no ill effects. The three strains of staphylococci were phage-typed by Professor G. S. Wilson and were all found to belong to the same phage type. This result was particularly useful in that the absence of clinical signs in the goat which was excreting *Staphylococcus aureus* made it doubtful whether the milk and cheese strains were identical.

There are also reports of staphylococcal food poisoning associated with spray-dried milk. Anderson and Stone (1956) record eight explosive outbreaks occurring in school canteens in England affecting 1,190 cases. The clinical features were characteristic of the toxin type of illness. No deaths occurred. The incriminated food was prepared from spray-dried skim milk powder. It was not subsequently heat-treated and was usually consumed 3–4 hours after preparation. The milk powder contained large numbers of *Staph. aureus* of a phage type often associated with food poisoning.

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Chapter X

FOOD HYGIENE

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In the widest sense 'food hygiene' comprises all those measures necessary to detect and prevent infection or intoxication. It is, however, conventional to confine this to specific measures aimed at providing the consumer with food which is both sound and safe; in particular, those measures directed at preventing or reducing hazards from bacterial food poisoning. Food hygiene also includes the various techniques necessary for the detection of disease in food derived from animals, e.g. meat inspection, sanitary design of premises, plant, utensils, apparatus, etc., hygienic control of practices and personal hygiene. These problems tend, in general, to grow geometrically rather than arithmetically as the scale of operations increases. Where the zone of operation is, for example, the family unit, a comparatively simple code suffices, but where communal feeding is concerned, standards must be more exacting.

In food-borne infection and intoxication man is almost always his own worst enemy. He will learn to live with the bacterial flora of the digestive or upper respiratory tract of other members of his family with whom he is in regular and intimate contact, but when subject to bombardment by 'foreign' organisms the result is frequently illness of greater or less consequence. Thus in an era when food habits have undergone radical change, as over the last decade or so in this country, food poisoning has shown a rapid increase. Over a long enough period, presumably, the majority of the populace would learn to adjust themselves to the 'foreign' invasion, but such a negative approach is not in consonance with modern concepts of social medicine, nor could one ignore the casualties during the period of adjustment or the minority who would always remain unduly susceptible. Moreover, most food poisoning arises from a series of unusual events, and control measures have, therefore, to anticipate the exceptional rather than provide just for the normal.

During a period of adaptation to new circumstances there will of course be occasions when gross neglect of elementary principles will occur and so contribute more instances of food poisoning than one ought to expect if common sense were to rule. Thus to attain

first a discipline suited to the normal circumstance should produce a substantial improvement; the exceptional will prove more intransigent, but by education of those concerned should eventually yield, so that ultimately food poisoning would be recognized to be as anti-social as any preventable illness. Enforcement has to play its part, for there is always the hard core which responds only to compulsion, but one has to rely for best results on intelligent co-operation, arising out of understanding of the problem and the reasons for measures adopted to combat it. The ultimate sanction after all must be informed public opinion.

Whether the problem be considered from the angle of education or legislation the crux of it may well have been summed up by Faulds (1948):

> After all, the standard of cleanliness that will satisfy a housewife, washing dishes for her family where they share the same tooth brush, hankie and cup, is not good enough for the public restaurant and should not satisfy the sanitary authority—yet that is the standard the dishwasher knows.

The common tooth brush must be rare by now, it might be remarked.

Consequently the provision of food which is both sound and safe requires an impeccable standard of premises, plant, and personnel wherever food is manufactured, prepared, stored, or distributed to the public. Minimum requirements can be laid down by law (e.g. Food Hygiene Regulations, 1955), detail of habits and processes by codes of practice; the need can be brought home by lectures and propaganda; but above all, frequent visits of inspection, as well as constant supervision of personnel are essential to emphasize, through personal contact with the man or woman actually on the job, the fundamental principles of personal and food hygiene. Old habits die hard and can only be successfully superseded when the need for new methods is understood and accepted. It may sometimes happen that techniques devised initially to serve the interests of food hygiene have an economic significance too and their adoption not only safeguards the health of the public but improves the efficiency of the industry. Conversely, changes in technique may have a food hygiene significance or, more frequently, some slight modifications may secure both objectives where otherwise one only might be served. Regular contact by frequent visits allows the inspectorate to seize or create opportunities whereby the 'sales appeal' of hygiene may be brought home on the jobs. Indeed, some of the major firms in the food

industry are finding it advantageous to engage their own hygiene officers.

Though no branch of food industry can ignore the principles of food and personal hygiene, detailed application will differ according to the type of food and nature of processing. Meat, milk, ice-cream, canning, preserving, for example, have all their particular problems. Records compiled from the Ministry of Health bulletins reporting food poisoning in the years 1951–5 show that of 813 general outbreaks (excluding those in hospitals), 615 occurred in canteens, restaurants, and institutions. The reports are careful to warn against attempting to incriminate any particular section of the food industry, especially as these figures relate to little more than a third of the total number, but it is reasonable to conclude that eating in public calls for greater safeguards than eating in private.

The large number of hospital outbreaks—267 in the same period—is in part due to cross-infection by salmonella organisms rather than to contamination of food; but at the same time the remainder illustrates the difficulties of controlling food-borne infections even when one has an enlightened personnel to deal with, trained in some degree in the practice of hygiene. Indeed, if one were to try to pin-point a single common factor at work in the increasing number of food-poisoning outbreaks reported, it would be a cause which is at the root of more ills than food poisoning—the loss of a sense of personal responsibility—and a remedy here would have to be sought in the realms of ethics rather than hygiene.

Food Handlers

The health, habits, and practices of persons engaged in handling food are of supreme importance. Coughs, colds, throat and ear infections, boils, skin diseases, surface injuries to hands, arms, face, gastro-intestinal disorders, infectious or contagious disease, are all potential sources of food-borne infection and intoxication. Persons so affected ought not to continue to handle food until fit again. Indeed, so far as these disorders fall under the definition of 'infection', food handlers (as defined in the Regulations) must notify their occurrence to the employer and he in turn, the local Medical Officer of Health, in compliance with the Food Hygiene Regulations, 1955, which deem protection of uninfected injuries by waterproof dressing to be sufficient safeguard. The Medical Officer of Health will decide whether an individual food carrier should be required under the Public Health (Infectious Disease)

Regulations 1953, to stop handling food. Even so an apparently healthy person can be a hazard to others if his or her habits while handling food permit the transmission of organisms from bowel or upper respiratory tract.

Cruickshank (1949) points out:

> Whilst food may be contaminated from many sources, man is undoubtedly the most important reservoir of the food poisoning bacteria. Even with the salmonella group whose natural habitat is the domestic animals and vermin, man is often the intermediary so that many more outbreaks of salmonella infection are traced directly to a human case or carrier than to an animal source. Since the bowel is the locus of most pathogens causing food-borne infection, the good social habit of washing the hands after use of the W.C. becomes an essential practice for the food handler. It may safely be claimed that if hand washing became as automatic as adjusting the dress after the use of the toilet, there would be a considerable reduction in intestinal infections.

Brice, Schested, and Dienst (1937) investigated the frequency of occurrence of faecal E-coli on the hands of food handlers in restaurants and cafés and ascertained its presence in 8·38 per cent of the 337 tests made, women giving a larger percentage of positives than men.

Horwood and Minch (1951) bacteriologically examined hand-washed samples derived from the hands of food handlers selected at random from 22 public eating establishments. They state:

> The large number of bacteria isolated from the hands of food handlers in this investigation and the frequency with which E-coli, haemolytic staphylococci and streptococci and aerobic spore formers were isolated indicate the magnitude of the problem of hand hygiene among handlers and the need for a greatly accentuated campaign of health education for this large and important group of workers.

The numbers and variety of bacteria from hands can, indeed, be turned to good account in health education by taking 'fingerprints' from handlers on agar plates. Incubated plates, after 48 hours or more, show convincingly the hazards which may lurk on even apparently clean hands. Similar demonstrations using hair, nail clippings, throat swabs, may profitably be employed—usually as 'visual aids' to a lecture or training course for food handlers, but where time and resources permit such demonstrations 'on the job' may prove even more valuable.

Asepsis is not practicable in the food industry, but measures can be taken to guard hands, and the food they will touch, from the more dangerous forms of contamination. Perhaps the greatest hazard results from necessary contact with the body during

performance of natural functions—hence the requirement of the Food Hygiene Regulations that 'Now wash your hands' notices should be displayed in association with sanitary and washing facilities provided in conformity with the Regulations. Nose-picking, nail-biting, smoking, snuff-taking, use of hand instead of handkerchief, are other habits which add substantially and dangerously to the bacterial flora of the hands; fondling and petting domestic animals are other reprehensible practices. The Food Hygiene Regulations make smoking and snuff-taking illegal while handling food (though some kinds of food business are exempt from the Regulations). Again, education rather than legislation is more likely to change habits or introduce safeguards, chiefly hand-washing after the hands have touched the body and before they again touch food. Unfortunately frequent hand-washing may lead to soft sodden skin which is more easily punctured and rendered unhealthy. Cruickshank (1949) recommends a hand lotion to keep the skin healthy and whole, the formula for which is as follows:

Powder of tragacanth	$1\frac{1}{2}$ fl. oz.
(43 ml. dissolved in a few ml. of alcohol)	
Oil of lavender	10 minims (0·6 ml.)
Oil of lemon	5 minims (0·3 ml.)
Glycerine	180 minims (11 ml.)
Water	to 1 pint (570 ml.)

The lotion may be thickened, if desired, by adding zinc oxide and calamine.

Code for Food Handlers

A code of conduct for food handlers, aimed at safeguarding the consumer, would include the following:

(1) Keep the body clean by washing and bathing. Hands should be washed at least on every occasion when entering food preparation or kitchen.

(2) Hands and arms should be washed thoroughly (using hot water, soap, and nail brush) after each visit to W.C. or urinal; remember toilet paper is porous.

(3) Avoid use of common (roller) towels or another person's towel. Paper towels (or tissues) or hot-air driers are preferable, or individual towels, frequently laundered.

(4) Wear a clean, washable overall (preferably white) or long coat with short sleeves. This is meant to protect food from the wearer's garments, not vice-versa. Cover the hair with turban or washable cap. Outdoor garments, hats, shoes,

should be deposited away from kitchens and preparing rooms.

(5) Keep hands away from nose, mouth, hair, eyes, during work.

(6) Do not handle unclean eating, drinking, or culinary articles.

(7) Do not wipe hands on overall or apron—use clean towel.

(8) Avoid contact with persons suffering from colds. Cover nose and mouth with handkerchief before sneezing (to reduce droplet infection) and do not shake out the handkerchief before use—especially over or near food.

(9) Use a spoon, dipper, ladle, fork, or tongs to serve or transfer food.

(10) Keep fingers away from rims and interiors of all clean drinking vessels. Do not lick the fingers to pick up paper; do not blow into paper bags.

(11) Wash hands and arms with hot water and soap after dressing or handling poultry, game or fish.

(12) No smoking, chewing, or snuff-taking in culinary departments.

Workers should be encouraged to adopt a positive approach by keeping themselves healthy; in particular emphasis is laid on the value of sensible footwear for those who are on their feet all day.

Medical Inspection of Food Handlers

Because of the risks from carriers of food-poisoning organisms it has often been advocated that workers in canteens, restaurants, food-processing, etc., should be subject to medical examination before being employed and that their continued employment should be conditional on re-examination at stated intervals. A physical examination would need to be supported by chest X-ray and bacteriological examination of stools and urine. Even so such a system means no more than that the person is fit at a particular time, and does not guarantee against contraction of infection shortly afterwards. Certificates of freedom from communicable disease are even less valuable. A false sense of security might be engendered by such a system which, to be of value, would require an exhaustive physical examination and a series of bacteriological negatives and thus be costly and time-consuming. Nevertheless some state and city ordinances in the United States prescribe medical examinations for food handlers and in this country some local education authorities require a clear chest X-ray and a negative stool before engaging staff in the school meals service. The Ministry of Education (Circular 272, 1954), recommends that

all applicants for employment in a school canteen should be asked to give their medical history. In any event employers would be wise to make necessary enquiries and adopt precautions before engaging persons to handle food. Certain industries have found it advisable for their own protection to adopt physical standards for entrants and the food industry might find such self-protection worth while without legal compulsion.

Food Premises

Rooms used as kitchens, preparation rooms, cleaning, stores, etc., should be well ventilated and provided with good natural and artificial light. For preference natural light should be through north-facing windows (or northern roof lights) of an area at least 1/10 floor space. There should be permanent ventilation to the outer air, supplemented by windows constructed to open; which should be protected by fly-screens. Use of anti-actinic glass and pale shades of blue for internal wall decoration also discourage flies, as does avoidance of direct sunlight by northern lighting (which also cuts down heat and glare). Fluorescent tubes are a satisfactory method of artificial light and can be corrected to give an amount and quality comparable with daylight. A suggested standard of illumination is that proposed in draft regulations for slaughter-houses: a general minimum intensity of 20 foot-candles rising to 50 foot-candles in areas where detailed work has to be carried out. Self-closing doors should be installed, outward opening to discourage entry of flies. General construction should be such as to be proof against insects, vermin, and rodents and prevent lodgement of dust, dirt, and flaking. Internal finish should also be such as to prevent condensation and include in general impervious surfaces, coving where two planes meet, avoidance of ledges, cornices, decorative work. Wherever possible the underside of roofs should be ceiled or otherwise treated so as to present an uninterrupted readily cleansed surface; service and waste-pipes should be similarly treated. A special precaution recommended by the Manufactured Meat Products Working Party (1950) is proofing against birds. Since this is aimed at excluding avian salmonellosis, all food-preparing rooms should be so proofed.

General layout should provide for separate food-preparing and vegetable-cleaning rooms (where these operations are carried out), refrigerated larder, dry store; within the rooms themselves apparatus and equipment should be arranged so as to facilitate an orderly sequence of operations. There should be ample space for

conduct of operations and circulation of personnel—even under conditions of maximum demand. Sanitary construction and layout must be supported by organization and management which maintain freedom from dirt, rodents, insects, etc.

Floors

In kitchens, preparing rooms, and sculleries an impervious but non-slip surface is necessary. Suitable materials include kiln-fired quarry tiles, cement terazzo, acid-resisting cement granolithic, rock asphalt. Hardwood, parquet, or sealed linoleum may be satisfactory in circumstances where grease, water, and organic fluids are not encountered. Duck-boards may be used around sinks, for the health and comfort of workers, but they must be kept clean by regular scrubbing. Floors must always be maintained in good repair, so as to prevent harbourage of dirt, insects, and rodents. Where large amounts of water are used, floors should be constructed with a fall either to a sufficient number of trapped gullies of adequate size, or to channels discharging externally over or into trapped gullies.

Drainage

No untrapped drain opening is permitted within a food room. Within a building branches should either be in lead-jointed cast (or spun) iron or concrete encased. Water-bearing floors of upper rooms must be bitumen sealed. Where one-pipe stacks are used, usual anti-siphonage precautions should be applied to traps.

Walls

Impervious surfaces should be provided to at least 6 feet or working height (whichever is greater). Suitable materials include glazed brickwork, tiles, terrazzo; cement (acid-resisting) treated with chlorinated rubber paint; plaster, similarly treated, provided it is in areas where damage to the surface is unlikely. Wood and plaster-board are unsuitable surfaces. Plastic or glass panels may be acceptable if adequately sealed against ingress of moisture, etc. Wall surfaces above working height should be painted. White is the colour most usually recommended, but other pale colours are acceptable. Pale blue, already mentioned as fly-repellant, has light-reflecting properties similar to white but is more restful to the eye and so provides greater working comfort.

Walls should be solid, not hollow, so as to prevent harbourage for rodents, insects, vermin, etc. Surfaces must be maintained in good repair and decoration.

Ceilings

A smooth surface free of depressions and projections should be constructed. Absorbent plaster, colour-washed, safeguards against condensation, but non-flaking materials must be used. Paint is liable to produce condensation, though rubberized matt finishes reduce the danger. Condensation is liable to cause contamination of food and utensils by 'drips'. Ventilating hoods and canopies fixed over cookers, boilers, etc., help to reduce condensation by disposing of vapours, gases, hot air, and odours. Mechanical extraction is a useful adjunct.

Services

Main services for power, heat, and water must be installed. Water must be potable, of adequate pressure to all appliances and adequate safeguards taken against cross-connection with non-potable supplies. A constant hot supply to sinks and wash-basins is necessary. Exposed pipes should be fixed clear of walls, horizontal pipes covered with insulating material, clear of the floor and painted. Lift-shaft casings should be smooth for easy cleaning and to prevent insect harbourage.

Food Stores

These rooms should have solid floors (tiles preferred), washable walls, solid ceilings. There should be good lighting, adequate air-space, and ventilation to allow free air circulation. Construction fly- and vermin-proof, particular attention being paid to proofing doors. Shelves should be 1-inch clear of walls for cleansing purposes; food containers, bins, etc., on racks 9–12 inches off the ground. Cheese should be stored in a perforated metal cabinet. Window ledges and other flat surfaces not intended for storage should be sloped so as to prevent their use for junk.

Separation of vegetable stores and vegetable preparing rooms has already been indicated. Rust-proof wire racks clear of the floor should be provided for vegetables.

General Maintenance

Sanitary construction and adequate size of premises are essentials to cleanliness and maintenance. There should be nothing

kept in food premises except equipment, utensils, and articles necessary for the work. Animals must be excluded (see Circ. M.-F. 20/51 of 24 October,1951).

Premises, fittings, equipment, utensils, etc., must be regularly and systematically cleansed. Special attention is necessary to positions which favour the collection of dust or dirt or harbouring of insects and vermin: namely behind or beneath apparatus and equipment fixed close to wall or floor, such as cookers, boilers, radiators, refrigerators, counters, and sinks. The value of cupboards and drawers for storage must be balanced against their misuse for discarded articles and rubbish.

Perishable Foods

An insulated refrigerated store is necessary for these items. Optimum temperature varies according to type of food; for example 36°–40°F. for fresh meats, 29°–32°F. for poultry and game, but never to exceed a maximum of 50°F.

Jensen (1945) recommends the following storage temperatures and humidities:

Product		*Temperature °F*	*R.H. per cent*
Fresh pork, pork sausage		26–28	75–85
Fresh beef cuts		33–36	80–85
Cooked meats		35–40	80–85
Boiled ham		26–34	70–80
Bacon, ham	Storage	26–28	70–80
	Display	38–50	70–80
Cheese		35–40	80–85
Vegetables		35–40	90–95
Fruits		35–40	80–90

Fruits and vegetables require thorough washing—scrubbing in some cases—before cooking. If used in salads or consumed raw scrupulous attention to cleanliness is imperative.

Ice

Ice for preservation or cooling should be manufactured from potable water, preferably on the premises as required. If storage is necessary, protection from human and animal contamination is essential. It should only be handled by tongs and washed before use under running mains water in a colander or sieve.

Storage Accommodation

Dry granular foods should be stored in receptacles provided with close-fitting lids to exclude insects or rodents; fly-proof wire gauze should protect meat. Food and drink exposed on counters

should be protected, by muslin or plastic covers, or glass cases, or within refrigerated display counters.

Disposal of Swill and Dry Refuse

Such material should be received in sanitary galvanized iron or steel refuse containers (preferably B.S.S.) provided with close-fitting lids, pedal-operated where possible to prevent contaminating the hands. Bins should be sited as far as possible away from culinary departments—swill bins outside on a raised cement platform with drainage to a trapped gully. A pressure hose or steam jet should be available for cleansing. Care should be taken to avoid spilling swill, as flies, etc., are thereby attracted. Bins, walls, and surround should, in summer months, be sprayed regularly with a suitable residual insecticide. Yards and areas should be regularly hosed, grids and gullies kept clean, and debris frequently removed.

Insecticides and Rodenticides

These must not be stored in culinary departments and their use in preparing rooms, kitchens, sculleries, etc., or where food, utensils, equipment, are used or kept should be strictly supervised so as to prevent contamination of food or equipment. Under no circumstances should baits of the so-called 'virus' type (i.e. incorporating bacterial cultures) be used in any food premises. Nor should cats or other animals be introduced on the pretext of rodent control. Rodent extermination must only be carried out by trained and experienced staff, with precautions against access of bait to food, equipment, utensils, etc.

Cloakroom Accommodation

Adequate cloakroom, w.c., and urinal accommodation must be provided for the staff and maintained in a cleanly condition. Wash-basins, supplied with constant hot and cold water, soap and towels should be sited strategically in regard to both w.c. and preparation rooms so that there is no excuse for failure to wash the hands.

Appliances, Utensils, and Equipment

It is preferable to site heating, cooking, and mixing appliances in a central position—certainly not less than 8 inches from walls and easy of access for dismantling and cleansing. Tables and

counters, etc., should have impervious joint-free surfaces; stainless steel or enamel tops are easiest to clean. Indeed, wood should not be accepted except for chopping blocks. Surfaces must be scrupulously and regularly cleaned— a wire brush being useful for chopping blocks. Tables, counters, etc., should not be less than 12 inches from the floor. Cupboards are necessary for storage of cooking utensils and partitioned drawers for cutlery. Trollies, trays, etc., used for transporting food must be constructed of suitable materials and kept scrupulously clean at all times.

Machinery used for slicing, mixing, mincing, etc., should be made so as to be dismantled easily for cleaning. This is best done by use of hot water, detergent, and stiff brush to remove food debris and grease, followed by a final rinse in boiling water. Ice-cream scoop or servers are to be cleansed in hypochlorite solution between each service, sterilized after use, and stored so as not to be contaminated. Other small items of equipment, palette knives, cutting knives, tin-openers, etc., must be washed frequently and especially at the end of the day's work being sterilized for 3 minutes in boiling water and afterwards stored so as not to be contaminated.

Cutlery, etc., should be of one-piece construction, stainless steel preferred, facilitating cleansing and sterilization. Special cutlery sterilizers may be installed for this purpose.

In general, all utensils, containers, equipment, apparatus used in preparation or service to be of impervious materials, designed to be free of crevices, ornamentation and projections which would trap food particles and make cleaning difficult. A similar simplicity of design and imperviousness of material are necessary for crockery. Chipped or cracked crocks, rusted, worn, corroded, or damaged metal, utensils, and equipment should be discarded.

Crockery Washing

The particular technique adopted will be governed by size of establishment and availability of labour. In small establishments manual methods will be used, but machines are generally installed where the turnover of crockery and saving of labour repay capital costs. In either case the first process is to remove food debris by scraping. A special table may be provided with a circular opening into a refuse container beneath. Scraping may be followed by pre-rinsing.

The ideal hand-washing method makes use of a three-sectional sink (or three separate sinks or tanks), the first containing

detergent solution, the second clean rinse water, and the third sterilizing solution. Detergent and sterilizer strength may be maintained by automatic dispenser, or strict control should be enforced to ensure that used solutions are emptied and new prepared at fixed intervals of time, calculated in relation to number of utensils cleansed and/or to temperature drop. In small plants the two-sink or tank method may be adopted, providing a detergent wash in the first sink and hot-water rinse (with or without a sterilizer) in the second. It is preferable that the constant-flow method be adopted.

Sinks should be constructed of glazed stoneware, suitable plastic or metal, designed so as to obviate angles, open seams, and crevices and also provide a smooth uninterrupted internal surface. Stainless steel, monel metal, iconel, or aluminium alloy are satisfactory metals. Any internal finish should be non-slip. Fine wire-mesh trays with handles are a useful adjunct. Sink wastes to be efficiently trapped and ventilated and mains water under pressure and constant hot water provided. Alternatively, galvanized or stainless steel tanks may be installed, heated by gas or electricity. A convenient working height is 3 feet to top of sink from floor level. Draining boards are best constructed of metal or plastic; hardwoods, such as teak, often favoured, harbour insects and are never absolutely impervious. Draining racks are best made of tubular, galvanized metal, and fitted with drip trays drained to sink waste.

Washing Procedure

(1) Scrape and pre-rinse.
(2) Immerse and thoroughly wash in first compartment in detergent solution at a temperature of 110°–120°F.
(3) Rinse or spray in second compartment in clean water of 110°F. temperature.
(4) Sterilize in third section, either by
 (*a*) Stacking rinsed articles in long-handled baskets submerged in clean water thermostatically maintained at a temperature of 170°F. for at least 30 seconds (some authorities recommend 180°F. for 2 minutes).
 or (*b*) Immersion for 2 minutes in clean water at 170°F. to which has been added a suitable chemical, e.g., hypochlorite solution (providing 100 p.p.m. free chlorine).
(5) Remove to draining racks to dry.

If brushes or cloths are used in cleansing they should be frequently sterilized by boiling. Glassware is best cleansed by use of brushes. There are sound bacteriological reasons against the use of drying cloths. Crockery, etc., sterilized at 170°–180°F. dries by evaporation, but if for any reason cloths are in use they must be sterilized by boiling before re-use. Plastic tableware retains insufficient heat to dry by evaporation and must be towelled. On no account should wet cloths be dried off and re-used. A hot-air cupboard is useful for drying considerable numbers. Clean crockery etc., to be stored under cover—for example in cupboards—and cutlery in covered metal containers.

Machine Washing

Mechanical washing has certain advantages, when the level of operation is high enough, which have already been indicated. Further than the economic case for their use, may be added that, since there is no contact with operators' hands, detergent strengths and temperatures can be increased. Indicating thermometers assist maintenance of proper conditions.

Scraping and pre-rinse precede mechanical washing. In the machine the order is similar to that for hand-washing, namely, detergent wash section (140°F. for 30–60 sec.), rinse section (170°–180°F. for 30–60 sec.); some machines provide also a section for sterilization (heat or chemical). Articles must be arranged in the machine so as to derive maximum advantage from spray-nozzles. Best results can be achieved where a number of machines can be installed each for a similar range of articles, since few machines can successfully deal with a wide range of shapes and sizes. Personnel must be adequately trained and instructed in the use of mechanical dish-washers.

Detergents

The physical basis of operation of detergents is that, by decrease of surface tension, soil is more readily removed by the water to which added, and by any 'mechanical' operations, such as agitation, use of squeegees, etc., in actual washing operations. A rather exacting specification is demanded of detergents suitable for use in the food industry: they should be cheap, economical in use, easy to handle, stable in storage, non-irritant to skin, non-toxic to man, leave no stain on china, earthenware, glass, metal, inactive with materials of which utensils are made, unaffected by

hardness or softness of water or by operating temperatures. A suitable detergent should be completely soluble, a good wetting agent, emulsify fats, deflocculate food solids, dissolve and remove quickly from plates, etc., be easily rinsed off in clean hot water,

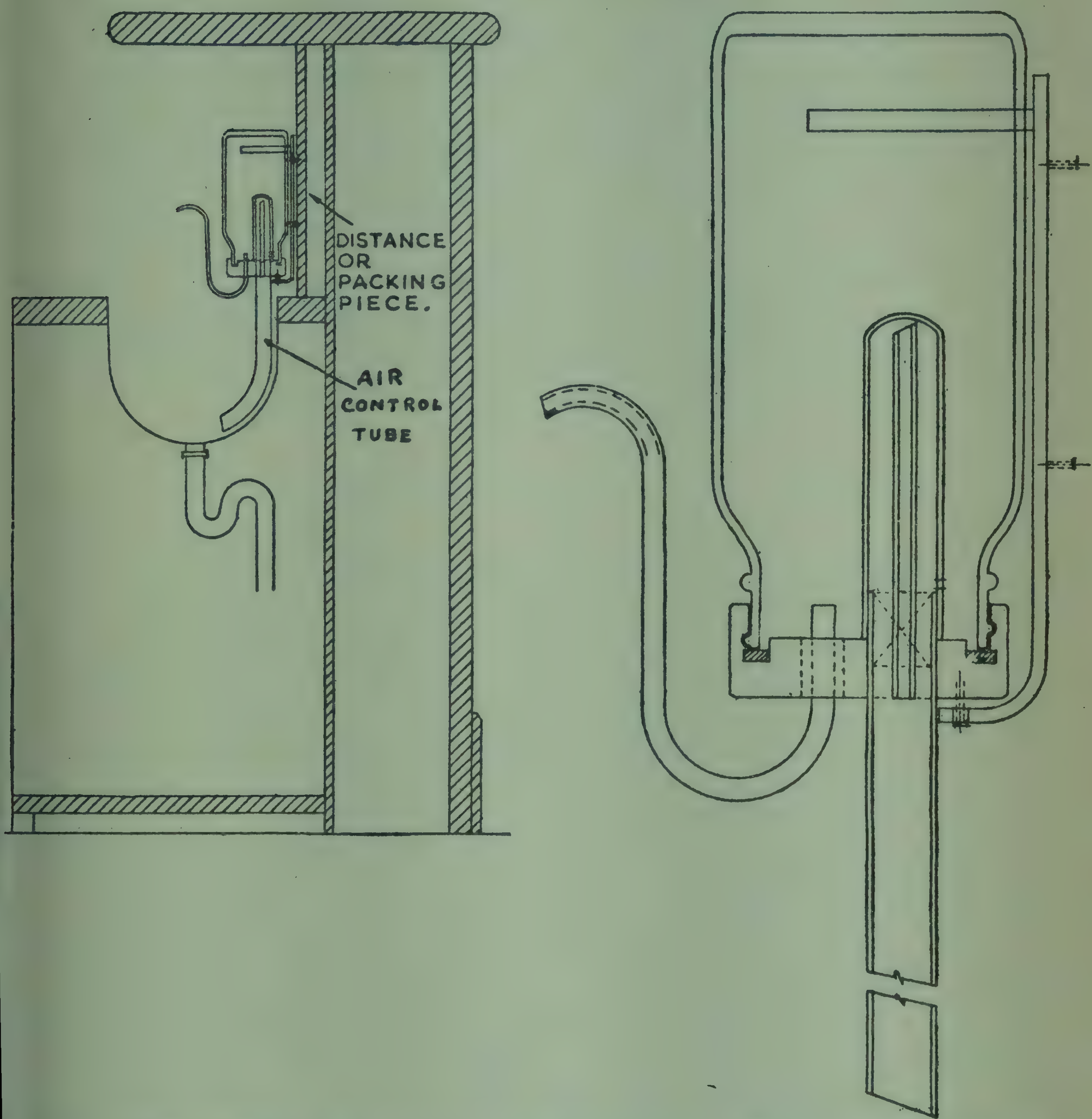

FIG. 3. TWO VIEWS OF AN AUTOMATIC DETERGENT DISPENSER (courtesy *Brewing Trade Review*).

leave a minimum of scale or scum and be non-poisonous in residual amounts remaining on food utensils. Precipitation of temporary hardening salts may cause a deposit on articles to be washed and sometimes combination with food debris.

Detergents in use include quaternary ammonium compounds, sodium silicates and phosphates, and cationic compounds. When used with chemical sterilizers it is important that they be

'matched' ionically, otherwise one may inactivate the other. Proprietary brands are usually paired so as to prevent this unwanted result. Inorganic detergents are usually a mixture of sodium hydroxide or sodium carbonate (useful for dissolving and degreasing), trisodium phosphate and sodium metasilicate (emulsifying), and sodium hexametaphosphate or similar phosphate (water softening and film inhibiting). Commercial detergents incorporate a suitable mixture.

Organic surface-acting agents include the sulphated alcohols which have useful wetting, emulsifying, and deflocculating properties but are unsuitable for use in mechanical dishwashers because of the amount of foam produced.

Use of chemical sterilizers and detergents in crockery washing have been investigated by Knox and Macdonald (1943), Knox and Walker (1947), and Frisby (1950), among others.

Testing Efficiency of Crockery Washing

The most important objective—to secure utensils not only clean but bacteriologically safe—can only be attained by understanding of the objectives of the exercise, by adequate staff training and by regular supervision, to ensure that at all times all that should be done is done. It is useful to be able to check the efficiency of operations from time to time. While physical evidence of retention of soil indicates that the operation has not been successful, absence of physical evidence may not always indicate success. It is also useful from time to time to have bacteriological confirmation, as by swabbing a sample of say half a dozen of each type of article washed, and by examining wash water samples. A useful test for these purposes is the M-D test for catering-utensil hygiene in Walters and Fowler (1950). Briefly a standard clinical cotton wool swab on a wooden applicator stick is moistened in bouillon and applied in prescribed fashion to each article. The swab is returned to the nutrient bouillon tube, stored in ice during transport to laboratory where samples are plated out. Examination should be not more than 2 hours after sampling. Results are expressed as total bacterial count per swab and as presumptive coliform. A satisfactory standard is less than 100 colonies per utensil, with no coliform. Wash water is sampled in a sterile 1 oz. bottle; if hypochlorite is used in the water, 1 ml. of a 2·5 per cent solution of sterile sodium thiosulphate is added to 20 ml. of wash water. Samples should be taken 15–30 minutes after washing up

commences. The standard suggested is not more than 500 colonies per ml. with no presumptive coli.

A supplementary physical examination may be performed by the use of finely powdered charcoal applied uniformly to crockery surfaces by means of a blower. No powder is retained by an absolutely clean utensil, but failure completely to remove grease etc. is shown by retention of fine dust. This test gives no indication of the bacteriological condition but can be of considerable educational value in indicating that scrupulous cleanliness is essential.

Legal Powers

These are contained in the Food and Drugs Act, 1955, Sec. 13; Food Hygiene Regulations, 1955; Model Byelaws, Series 1; Shops Act, 1950, Sec. 38; Public Health (Infectious Disease) Regulations, 1953.

Hygiene of Alcoholic and Soft Drink Services

Non-alcoholic beverages and beer have from time to time been implicated as vehicles of chemical food poisoning, usually as a result of unsuitable materials of which vessels, pipes, etc., have been constructed. Milk has been known to convey bacterial food poisoning. In most of the cases recorded it has been found that contamination has occurred at some stage earlier than the dispensing of the beverage and this aspect of food hygiene has tended, therefore, to receive minor attention. Absence of recorded cases of food poisoning cannot, however, be a reason for non-observance of hygienic principles, and the serving of beverages, alcoholic or otherwise, for immediate consumption presents hazards to communal health which need not be elaborated. Some of the complacency with which the licensed trade has, until recently, viewed these problems is based on properties of their main stock—beer—which have been held to provide safeguards lacking from other beverages. Since beer is spoiled unless its microflora are controlled, a quasi-hygienic procedure is normally followed at the brewery and in the cellars for economic reasons.

Bunker (1948) recommends the following hygienic precautions:

Plant and Utensils

(*a*) *Mains* should be regularly cleansed by detergent and clean rinse.
(*b*) *Coolers* should be cleansed by weak caustic solution and then thoroughly washed.
(*c*) *Open vessels* according to materials e.g. glass and stainless steel linings can be sterilised after washing, aluminium can be cleaned

with soap solution and glass-wool; buckets, cans, scoops, treated with caustic solution, rubber hoses with weak sulphite or peroxide—all these chemical treatments must be followed by clean rinse.

Fermenting vessels require special treatment to remove ' beer stone ', e.g. a cream of caustic soda and china clay for their deposits, acid treatment if they are heavy.

Bottles, fillers and casks. Mechanical bottle-washing is adaptable to the brewery industry, though the detergent must be selected with care to avoid adverse effects on the beer. Fillers should be cleansed with caustics and phosphates—the general principles of dairy hygiene would apply here—and casks cleaned with hot water (140°F. for waxed-lined casks) or steam (for untreated casks).

Cellars and Floors must be regularly hosed and treated periodically with chloride of lime, antiform or chloramine.

The *p*H of beer, usually around 4, the preservative effect of soft resins of hops and the small amount of alcohol present, also help to control the bacterial content, so that spore-formers and pathogens are rarely found. These properties have also been thought to offer a safeguard against contamination of glasses, tankards, etc., from the lips of drinkers. Observers have shown, however, that such optimism is illusory.

Shimwell (1950) found that gram-negative bacteria withstood the action of hop antiseptics. Iddison (1952) records non-haemolytic streptococci and/or coliform bacilli in 15 out of 18 samples of overspill taken in Dartford and quotes reports from Crapper of E-coli and staphylococci in overspill. Further evidence of contamination of drinking vessels during use is provided by Davis and Resuggan (1947) who record counts in rinse water from 10,000 per c.c. to 'uncountable', coliform present in from 1 ml. to 1/10 ml., and counts of swabs from glasses from 5,910 to 227,600. Bunker (1950, 1952), Tyler (1950), and the Report of the Catering Trade Working Party (1951), add further evidence of the need for thorough cleansing and sterilization of all drinking vessels.

In spite of this, it is often found that the only provision for cleaning glasses is a bowl of cold water beneath the bar counter or perhaps a small sink supplied with cold water and wastepipe discharging into a bucket. This is unsatisfactory from a hygienic point of view. There may be circumstances where the provision of the two or three-sink system of washing and sterilization can be made, but often limitation of space and the demands of rush-hour periods where glasses are demanded in excess of supply, call for the provision of a more simple but sure method of dealing with these drinking vessels.

Methods of Cleaning Glassware

Sinks should be of sanitary construction and materials, efficiently trapped and ventilated (if necessary). Stainless steel with metal drainers is recommended; wooden sinks and draining boards are objectionable from a sanitary point of view. Potable cold water under pressure and constant hot water must be provided over the sink. Where glass-washing can be separated from bar service it should be possible to provide a two-section sink. In the first section water should be maintained at a temperature of 110°–120°F., with automatic addition of detergent to maintain correct concentration, the detergent wash to be changed frequently in busy periods at 30-minute intervals. After cleansing, the glasses must be immersed for 30 seconds in clean water at 170°F. in the second sink, to effect sterilization. The sterile glasses are inverted on the drainers to dry and then placed inverted on clean shelves. Ideally, the glasses should not be towelled but polished glasses are usually considered a 'must' in these branches of the catering trade. When cloths are used they must be frequently changed and washed at the end of each day in hot detergent solution, wrung out and dried. The drainers should be swabbed from time to time throughout washing operations, using cloths soaked in detergent solution made up especially for the purpose and not dipped in the detergent wash in the sink. This procedure also applies to counter swabs.

Where space and speed of service preclude the provision of the two-section sink, the minimum acceptable is a single sink and drainer with hot and cold supply and automatic detergent dispenser. Since hand-washing only limits temperatures to 110°–120°F., glasses will not effectively dry without towelling, so that the precautions already recommended for drying cloths must be strictly observed. Change of detergent-wash at 30-minute intervals must be rigorously maintained.

Glass-Washing Machines

Capital cost and space are the main objections to mechanical glass-washers. Bunker (1950) criticizes the insufficiency of speed of operation. Mechanical breakdowns must be avoided by adequate maintenance, but it is always a possibility, so that a secondary means of glass-washing has still to be available in emergency. Chief points to be observed are that means be adopted to maintain the water temperature at 170°–180°F., that the detergent solution

is kept at the correct strength and that a minimum period of immersion of 30 seconds is achieved.

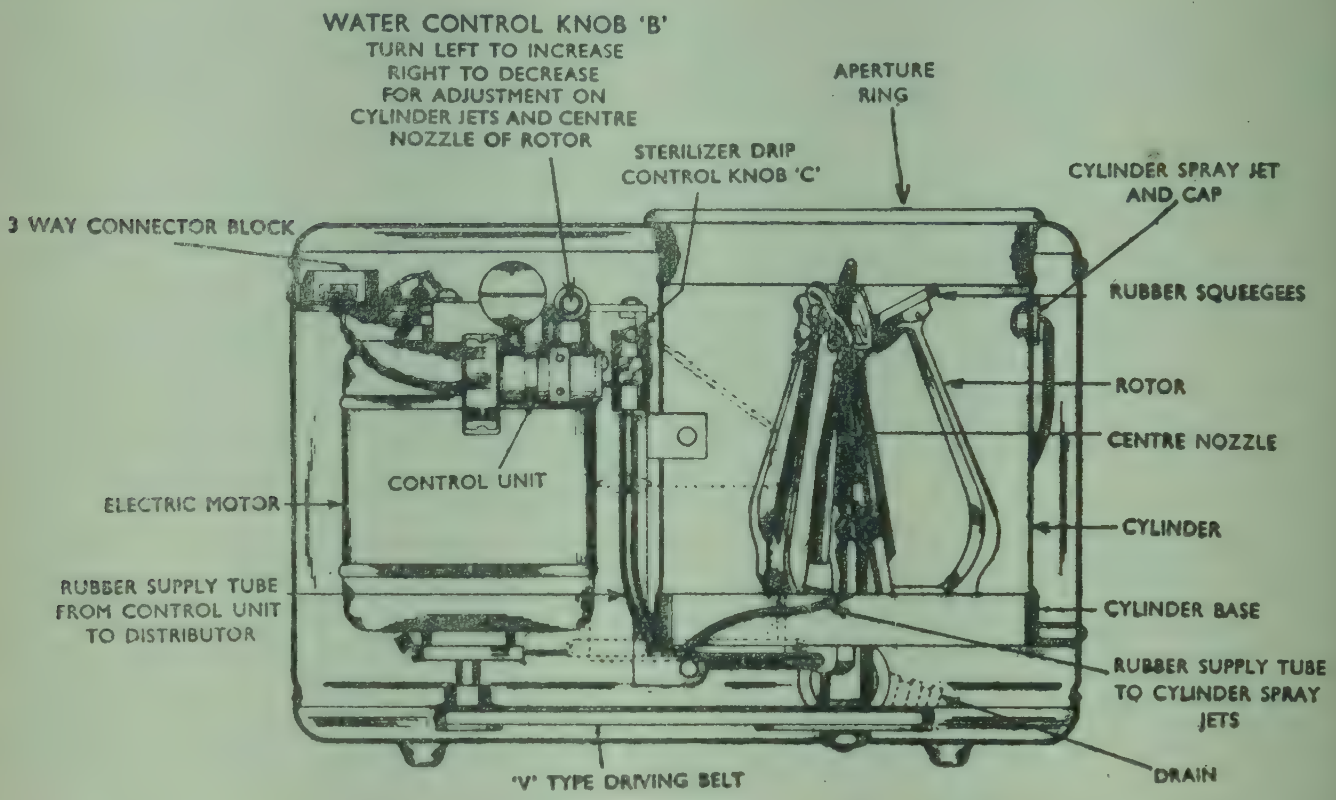

FIG. 4. GLASS-WASHING MACHINE (courtesy *Moreton Engineering Co. Ltd.*)

DETERGENTS

The general properties required of detergents to be used in cleansing food utensils and equipment have already been specified. Those for use in the hygienic drink service should in addition have a rapid and non-selective bactericidal action, be odourless, tasteless, non-toxic, non-irritant to the skin, and economical in use. They should leave no film or slipperiness on the articles treated with no haze-forming properties and no deleterious effect on beer-head. Quaternary ammonium compounds meet these requirements and various proprietary formulations of alkyl pyridinium are used in automatic dispersers. It is important to follow manufacturers' instructions for use. Care must be taken not to mix 'paired' products; for example, detergents recommended for use with quaternary ammonium compounds are likely to inactivate hypochlorites and vice versa. Manufacturers do not merely recommend a detergent-sterilizer combination to boost their own sales—there are sound chemical reasons for using matched products. Quaternary ammonium compounds have particular value for cleansing drinking vessels in that there is a 'residual' action and thus a considerable abuse (as by prolonging the period between changes of detergent wash) can be tolerated in emergency.

Sterilizing Solutions

Although steam and boiling water are ideal for sterilization, their use for glassware is not recommended because of the risk of fracture. Plastics cannot usually be subjected to sterilization temperatures. A simple and inexpensive sterilization procedure is the addition of chlorine to rinse-water. Mallmann and Devereaux (1935) found 200 p.p.m. available chlorine to be effective. Stable formulations of sodium hypochlorite are the most convenient method of chlorination.

Efficacy of cleansing and sterilizing techniques can be checked by methods described earlier for catering utensils.

A photometric method of assessing detergent efficiency is described by Mann and Ruchoft (1946) in the Report of the United States Public Health Service.

Beer Engines and Pipelines

Construction, maintenance, and methods of cleansing apparatus used for raising beer from the cellars must receive attention if contamination is not to occur. Use of lead alone, or in combination with other metals, in the construction of pipelines is likely to result in contamination. Tyler (1950) records use in 25 per cent of pipelines in Bath and excess lead in 62 per cent of samples of beer. McDonald and Yates (1943) in Leicester found lead alone or combined in 38 per cent of pipelines. Risk is greatest when 'first draw' is pulled and regular sampling for metallic contamination should be practised where pipelines are constructed of lead or lead alloys.

Glass pipes may be used, though risk of fracture is a disadvantage; beer can be seen in transit from barrel to pump. Translucent plastics are a more recent development and are not likely to be broken. Stainless steel, with universal joints, is most generally favoured. Telescopic joints should be avoided. Regular dismantling for cleansing with detergent is necessary—preferably weekly, but never less often than monthly.

Cellars

These should be kept clean, adequately lighted, and ventilated. Floors should be of impervious construction drained to a trapped gully, the seal of which should be periodically checked. Walls and ceilings should be of sanitary construction and the whole structure maintained in good repair. Refuse must not be allowed

to accumulate and regular cleansing of walls, ceilings, and hosing of floors must be practised.

Wine cellars may present a problem in infestation control because moth larvae may attack corks and fruit flies may breed in the resultant spillage. Microbiological control of wine-making provides a substantial safeguard against health hazards.

The hygienic service of wines, spirits, soft drinks follows the recommendations for licensed premises. Dispensing of 'still' beverages in single-service containers is a commendable procedure provided hygienic storage of containers is assured.

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PART III

Chapter XI

CONTAMINATION OF FOODS BY POISONOUS METALLIC SALTS

Chemical poisoning is comparatively rare in this country, but the salts of poisonous metals do occasionally find their way into foodstuffs.

Normally, vegetables and animal foods contain minute traces of many elements, and analyses have proved that at times such metals as copper, arsenic, iron, etc., are found therein, but usually only in very small amounts. Chapman and Lindon (1926) proved the presence of arsenic and lead in marine crustaceans and shellfish, and they came to the conclusion that the metals were derived from the sea water. Samples collected from the Thames and Medway each averaged about $\frac{1}{40}$ gr. of arsenic per gallon (0·33 p.p.m.).

The eating of certain fish, such as plaice, which may contain this metal up to 3 p.p.m. leads to the presence of quantities of arsenic in the urine within 24 hours; but whether such forms of arsenic and lead are really poisonous to human beings is a matter for investigation.

The rôle which metals play in food is a complicated one. It is a well-known fact that they may combine with the protein, thus neutralizing any poisoning effect and rendering the food more or less harmless, except where the metallic salts are present in excessive amounts. This probably is the reason why some individuals, exposed to metallic poisoning, show no symptoms and are able to ingest and eliminate quite large amounts of metal which may cause illness to others. As a rule cases of food poisoning caused by the ingestion of food contaminated by poisonous metallic salts have an onset of symptoms immediately after the food is eaten. Vomiting, nausea, and pain are the usual symptoms and these may occur from 2 minutes up to 2 hours. The severity of the symptoms depends to a large extent on the resistance or idiosyncrasy of the individual and the amount of the metallic poison consumed. A large amount induces immediate vomiting which may cause complete expulsion of the poison. A smaller quantity may not cause vomiting but only gastro-intestinal disturbance.

Savage (1941) referring to investigations of chemical poisoning, remarks:

> The history of the outbreak, the very rapid onset after consumption in the acute cases, and the characteristic metallic poisoning symptoms usually puts the investigator quickly on the track.

Owing to the widespread use of metals in the food industry many manufactured products, during preparation, come into close contact with machinery and containers during cooking, processing, storage, transportation, and distribution, and there appears to be little doubt from the work of analysts that a certain amount of metallic contamination does occur, the degree usually depending on the length of time the foodstuff remains in contact with the metal. As a result of modern chemical and biological researches, manufacturers are now realizing the great importance of the precautions that must be taken during the handling of all such apparatus, especially the limitations to be placed upon the storage of foodstuffs in contact with metals. Moreover, these researches enable selection of suitable equipment to be made for use in factories and other places where food is manufactured, prepared, and stored. Tanks and other apparatus lined with glass, or other special materials, not subject to ordinary corrosion by the product which may be brought into contact with them, have been introduced, thus reducing metallic contamination to a minimum.

Increased public interest has affected the general attitude towards foods. Manufacturers appreciate the necessity for hygienic methods in preparing and handling foodstuffs and the importance of protecting them from contamination so that they may reach the consumer in the best possible condition.

Canned Food

Formerly, cans were made by hand and solder was used for sealing the top, sides, and bottom. Thus metallic contamination was likely to take place. In the manufacture of modern cans, however, no solder comes into contact with the contents; it is only applied on the outside, the ends being put on by means of a metal-to-metal seam with a thin layer of rubber compound between, the effect of which is to prevent contact with any lead from the solder, so that the only contamination that can take place is from the tin and iron which is practically nil.

During the process of tinning it is not possible to obtain an absolutely perfect coating on the steel sheets (the amount of tin does not usually exceed 2 per cent); consequently precautions have

to be taken to prevent interaction between the containers and the contents. The cans are lacquered to obviate any corrosion and to avoid change of colour in the food. Results of investigations carried out at the Campden Research Station, however, proved that this small quantity of tin is removed during the first two months of storing.

Buchanan and Schryver (1908) in their report to the Local Government Board, stated that

> practically all foods canned in the ordinary way become to some extent contaminated with tin as a result of the contact of the food with the tin-plate of the can. Tin is taken up by meat extracts and essences to a greater extent than by most other meat foods. This results from the acidity naturally possessed by the meat extractives in these preparations. Certain canned fruits and vegetables, and foods such as canned soups, of which the latter form part, are also specially liable to take up tin from the can in consequence of their natural acidity. In such cases, tin may penetrate into the substance of solid foods, and in the case of canned foods, which consist of both liquid and solid portions, e.g. canned fruit, the solid portion may come to contain relatively larger proportions of tin than the liquid. This results from the fact that the tin, after solution in the liquid contents of the can, becomes in course of time absorbed to, or chemically combined with, the solid contents.

In some canned meat and fish products, protection from contact, with the can and subsequent discoloration is obviated by using paper liners.

Contamination of canned fruits and vegetables by tin has been thoroughly investigated at the Campden Research Station. The results of the experiments go to show that certain vegetables are liable to remove more tin from the surface of the container than do acid fruits, and it is recommended that the inner surface of the can be protected by a lacquer. Very encouraging results were obtained from cans in which the second coat of lacquer is sprayed on after the tins are made up, and thus any scratches in the first coat of lacquer which exposes the iron are covered by the second coat.

In 1934 it was recommended that one method for obtaining improved protection was to spray the interior of cans made from twice-lacquered plate with a third coat of a quick-stoving lacquer. Trials with English fruits, which normally give trouble, were carried out on these lines with decidedly advantageous results.

In the Food Investigation Special Report No. 44, 1936, it is suggested that in all probability the corrosion of cans by foodstuffs will eventually be overcome by improvements in lacquers and methods of lacquering. Failing such a development, relief must be sought through improvements in the tin coating in the steel base,

in the cold storage of canned goods, and in the application of knowledge concerning the corrosion of tin-plate. The metals usually associated with the contamination of food are: arsenic, antimony, copper, lead, aluminium, tin, and zinc.

Tanner (1933) states that metallic salts may reach foods in different ways, as follows:

(1) They may reach the food by accidental mixing of the metal or its salts, as illustrated by contamination of sugar with arsenicals during shipment, preparation of foods in containers of unknown origin, etc.

(2) They may reach the food by solution from utensils in which it has been handled or processed. Such agents which might add a deleterious metallic salt have, in the main, been eliminated.

(3) They may be added to the food for some special purpose. The use of lead arsenate sprays for destroying insects on fruits and vegetables is a good example.

(4) They may be naturally present. Some foods, such as marine products, contain appreciable contents of metals. Such metal is apparently bound with proteins in the food and is not available until released, to poison the tissue.

Arsenic

Probably no metallic contamination of food is of so much interest or importance as that of arsenic. It is present in sea water and the soil, thus gaining access to both animal and vegetable products which go to make up the human diet. Williams and Whetstone (1940) records 0·3 to 38 p.p.m. of arsenic in normal soils. It is present in the human body, is excreted in the urine, and traces may be found in the nails and hair. Arsenic often occurs as an impurity in many chemicals which are used in one way or another in the food industry, consequently it is easy to understand that contamination of food at times is liable to take place. Traces of the metal have been found in jams, sweets (Hutchinson, 1909–10), lemonade, liqueurs, sugar, marmalade (Rupp, 1908), treacle, and syrups, some of which commodities are largely manufactured from glucose. The use of glucose as an admixture or an adulterant is open to serious objection, unless it is known to have been prepared with acid freed from any arsenical impurity. At one period it was common for sweets, etc., to be coloured with arsenical pigments, but under the Public Health (Preservatives, etc., in Food) Regulations of 1925, the use of metallic colouring matters and compounds of certain metals in food is forbidden.

Arsenic has been the cause of food poisoning on several occasions, owing to the fact that this substance is tasteless, odourless and cannot be detected ordinarily when mixed with foodstuffs. It

has at times become accidentally mixed with, and mistaken for, flour or sugar with serious results. The following outbreak of arsenical poisoning occurred in Vienna. An apple-cake was 'sugared' by mistake with arsenic. Two persons who consumed some of the cake died, whilst others were seriously ill with vomiting and diarrhoea. One case showed in addition a severe swelling of the conjunctivae; another an exanthema; a third acute laryngitis with aphonia. One of the most notable outbreaks was in 1900 at Lancashire, Cheshire, and Staffordshire, where some 600 persons were poisoned by the presence of arsenic in beer, 70 of the cases proving fatal (Reynolds, 1901).

The Royal Commission appointed to investigate the outbreak recommended that the arsenic content of substances used in food manufacture should not be greater than $\frac{1}{100}$ gr. per lb. (= 1·4 p.p.m.) for solids and $\frac{1}{100}$ gr. per gallon for liquids (=0·14 p.p.m.), the arsenic being expressed in terms of the oxide As_2O_3. Wynter Blyth states, 'the smallest single dose of solid arsenic said to have proved fatal to a human being is 0·16 gm. ($2\frac{1}{2}$ gr.).'

The Metallic Contamination Sub-Committee appointed by the Ministry of Food in July 1948 to undertake an investigation into the contamination of foods with metals or other injurious elements recommended the following revised limits for the arsenical contamination of foods:

For beverages ready to drink a limit of 0·1 p.p.m. As (0·13 p.p.m. As_2O_3) and for other foods 1·0 p.p.m. As (1·3 p.p.m. As_2O_3).
Foods to which the general limits cannot at present be applied are mainly concentrated foods or food adjuncts.
The limits recommended are given in the table on p. 192.

Calvery (1941), discussing trace elements (arsenic) in foods, remarks:

When administered to both animals and men, even in its most insoluble forms, it is soluble in the secretions of the gastro-intestinal tract and is absorbed, some of it being stored in the tissues, but the greater portion being excreted in the urine. The storage is principally in liver, spleen, muscle, skin, hair, and brain, and that stored in the brain tissue remains more constant over a period of time than that stored in the tissues of other vital organs. As a result of the storage of arsenic in the tissues of the body, conditions directly attributable to it, namely, pigmentation, dermatitis, exfoliation, neuritis, hyperkeratosis, and various forms of cancerous conditions of the skin, have been observed years later. There is wide individual variation in the physiological response to arsenic in both man and animals. Some persons become sensitised after exposure to it so that subsequent exposures produce more marked toxic manifestations than the first exposure.

The principal manifestations of arsenic poisoning are gastro-intestinal disturbances. In acute poisoning, after the ingestion of large amounts of arsenic, the symptoms are violent gastro-enteritis, pain, vomiting, watery or bloody diarrhoea with threads of mucus;

Article of Food	*Parts per million by weight* *Arsenic*	*Arsenious Oxide* (As_2O_3)
(I) BEVERAGES:		
Cider	0·2	0·26
Black beer	0·5	0·66
Soft drinks intended for consumption after dilution, concentrates used in the manufacture of soft drinks and undiluted fruit juices	0·5	0·66
(II) OTHER FOODS:		
Ice-cream, iced lollies and similar frozen confections	0·5	0·66
Dehydrated onions; Dried hops; Dried liquorice extract; Edible gelatin (already prescribed); Liquid pectin; Chemicals (other than chalk) used as ingredients or in the preparation or processing of foods	2·0	2·6
Chalk (creta)	4·0	5·3
Chicory—dried or roasted	4·0	5·3
Dried herbs; Finings and clearing agents; Hop concentrates; Solid pectin—all grades; Spices	5·0	6·6
Food colourings other than synthetic colourings	5·0 on dry colouring matter	6·6 on dry colouring matter
Water and milk should not contain more than 0·1 p.p.m. of arsenic (As).		

later, skin cold and clammy, blood pressure falls with marked weakness. Convulsions and coma precede death, which is due to circulatory failure. Small and repeated doses of arsenic cause restlessness, nausea, vomiting, headache, dizziness, chills, cramps, irritability, and progressive paralysis.

THE SPRAYING OF FRUITS AND VEGETABLES WITH POISONOUS INSECTICIDES

From time to time arsenic has been found in excessive amounts in the wrappings and skins of imported pears. In one case $\frac{2}{3}$ gr. per

lb. was present in the wrappings and $\frac{1}{12}$ gr. per lb. in the skins. In 1926 samples of apples were taken for analysis. Five of the samples of Jonathan apples imported from America were found to contain arsenic, in each case more than $\frac{1}{100}$ gr. per lb. Samples of English apples were found to be free from the metal. Again the results of investigations on English, Canadian, and American apples showed 11 of 24 samples to be free from arsenic, 9 contained traces, and 4 appreciable amounts.

During the spraying of fruit trees with arsenical insecticide, the soil beneath may become heavily contaminated.

Tanner (1933) says:

> In several quarters the widespread use of metals and their salts in the foods industries is viewed with alarm. Myers and Throne (1929) have recently pointed out that the public at large is submitted to the same action of arsenic as are the insects on sprayed fruits. They pointed out that arsenic is passing into the circulatory system as evidenced by its secretion after eating food which contain it. They claimed that too little attention is given to arsenic in foods and that imperfect fruit would be preferable to contaminated fruit.

Suggestions have been put forward from time to time regarding methods for the removal of poisonous residue on fruits caused by spraying with insecticides. It has been found that brushing and wiping will not remove arsenical compounds; but the action of hydrochloric acid on lead arsenate is a solvent one. By careful flotation of the apples in a 0·5 to 1 per cent solution of the acid at a temperature of 80°–90°F. followed by thorough washing in running water, its removal can be effected. Any increase in the strength of the acid solution or the temperature would probably injure the apples.

It has been suggested that heated solutions of sodium silicate may be found quite effective for removing the residue from apples that have been sprayed with lead arsenate insecticide. In the United States the Department of Agriculture has devoted much attention to the subject of fruit insecticides and established regulatory tolerances for arsenic, lead and fluorine as follows: 0·01 gr. arsenic as arsenic trioxide, 0·01 fluorine and 0·018 lead, per pound of fruit. (Farmers' Bulletin U.S.A. No. 1752, 1935–43, and Further Studies on the removal of Spray Residues from Eastern-grown Apples, Department of Agriculture, 1942, Tech. Bull. No. 828.)

Antimony

The salts of this metal, which is widely distributed in nature and a powerful poison, are seldom a cause of food poisoning.

Fairhall and Hyslop (1947) record:

> Some knowledge of the physiological effect of antimony existed in ancient times and is typified by use of the pocula emetica or calices vomitorii of the Romans. These goblets, consisting of an alloy rich in antimony, were used to provoke emesis by drinking wine which had been allowed to stand in them for a few days. The amount of antimony dissolved was sufficient to produce vomiting. During the middle ages, the physiological action of antimony engaged the interest of many individuals, particularly the iatrochemists and various compounds were employed in medicinal preparations.

Several outbreaks, however, have been recorded both at home and abroad, due to cheap enamel-coated utensils which yielded up a sufficient quantity of soluble antimony to cause serious illness.

Lehmann (1902) drew attention to the dangers of antimony dissolved from enamel by vinegar and other acid foods.

Järvinen (1925) also found that appreciable amounts of antimony dissolved from certain brands of antimony enamel-ware and Flury (1927) pointed out that, since antimony-enamel-coated cooking utensils give off small amounts of antimony in cooking, this type of enamel should be replaced by an enamel containing nontoxic material. In criticism of this statement Rewals (1924) stressed the nontoxic character of the pentevalent compounds of antimony. Outbreaks have occurred in this country, in Newcastle-on-Tyne (Dunn, 1928), Folkestone (Monier-Williams, 1929) and in London (Monier-Williams, 1932), and were caused by lemonade made from lemons or lemonade crystals which had been prepared or stored in enamelled jugs or pails. The citric or tartaric acid dissolved out dangerous quantities of antimony from the utensils causing illness in a large number of persons in each outbreak.

In the outbreak which occurred at a London hospital in 1932 during a nurses' Christmas dinner, 65 persons were seized with acute vomiting, followed in some cases by collapse. The cause was found to be the lemonade which had been prepared from lemons in white enamelled jugs.

It would appear that during recent years antimony oxide being cheap has been widely used as an opacifying agent in place of tin oxide. Antimony pentoxide, while safe as an enamel ingredient, owing to its very slight solubility in acids, becomes reduced during enamelling to the poisonous trioxide which is easily soluble. The remedy is to have an enamel matrix insoluble in acids because of a sufficiency of silica which encloses the oxide particles.

The Ministry of Health (1933) issued a memorandum in which attention was drawn to the possible danger of antimony due to the

use of enamelled vessels with acid drink, like lemonade. In 1934 a pamphlet (No. 73) by Monier-Williams on 'Antimony in Enamelled Hollow-ware' was also issued by the Ministry of Health, in which he summed up the situation as follows:

> The recent outbreaks of poisoning are attributable to the presence of relatively large amounts of antimony trioxide in enamels which were relatively low in silica and therefore soluble. The enamel matrix was disintegrated by the acid and the antimony oxide dissolved. There is reason to believe that almost all enamels containing antimony give up small amounts of the metal to food, and it is at least questionable whether the continued ingestion of these small amounts may not be injurious to health. It is suggested that total prohibition of antimony in hollow-ware might be found to be in the best interests both of the public and of the trade.

Burns (1935) carried out a number of experiments as to the effect of citric acid solutions on enamel-ware. His summary is as follows:

> (1) Antimony compounds are extracted from 'hard' enamels by the action of dilute citric acid solutions.
>
> (2) Cleaning and scouring of enamel vessels expose a new surface to the action of the citric acid. With consequent increase in the amount of antimony extracted.
>
> (3) Storage of citric acid solutions in enamel-ware is shown to be dangerous, owing to the rapid increase of the antimony-content of the stored solution.
>
> (4) A slightly modified method is given for determining the antimony-content in such solutions.

See also Coste and Garratt (1935).

Bleyer and Spiegelberg (1933) found that beer and wine take up considerable amounts of antimony from rubber tubing, as the latter contains antimony pentasulphide. Donovan (1934), investigating this matter, confirmed that beer in passing through red rubber hose contained antimony. He considered, however, that risk from poisoning was slight but that such rubber should not be used for conveying beer.

Poisoning from the ingestion of food contaminated with antimonial salts is usually of a mild character and of short duration. The symptoms which occur in a few minutes to 1 hour are characterized by headache, nausea, vomiting, gastric pain, and general lassitude. Diarrhoea is usually absent. In chronic cases, where antimonial salts have been absorbed slowly into the system, there is dryness of the throat, pain in swallowing, persistent nausea, occasional vomiting, loss of appetite and weight, muscular

weakness, giddiness and diarrhoea with the typical 'rice water' stools, followed by cramp. The urine may contain albumin and blood. Death generally results from collapse. Antimony is excreted mainly by the faeces, but traces can be detected in the urine, bile, sweat, and milk.

Manley (1930) found 17 p.p.m. antimony in Gruyère cheese which had been wrapped in tin foil, the latter containing 3·2 per cent of antimony.

During 1934 an unusual case of contamination by antimony occurred in Japanese canned oranges. Some of the samples contained 0·14 gr. of antimony per lb. (corresponding to 0·37 gr. of tartar emetic). It was suggested, however, that the oranges became contaminated by being prepared in vessels coated with a soft antimony enamel.

The medicinal dose of a soluble antimonial salt should not exceed 1½ gr. A fatal dose (2 gr. or more) may cause death within a few hours to a few days.

Cadmium

This is a silvery-white soft metal, which is used extensively (especially in the United States) for plating utensils, fittings for electric cookers, refrigerating apparatus, and washing machines, etc. Cadmium is also utilized in combination with other metals such as silver, lead, tin, and copper, as a soft solder. Cadmium-plated articles have a highly polished surface and this affords protection against rust. The metal is, however, readily attacked by some acids (sometimes by acid foods), and drinks such as lemonade, fruit juices, and wines, but it is insoluble in alkalis.

The salts of cadmium are poisonous, consequently the plating should not come into contact with beverages or foodstuffs. Cases of poisoning have been recorded from time to time in this country and in the United States due to contaminated food or drink. Taylor and Hamence (1942) reported 8 cases of poisoning from cadmium caused by the consumption of lemon ices prepared in plated refrigerator trays. Ices prepared in a similiar manner contained 190 to 280 p.p.m. of the metal (see also Frant and Kleeman, 1941). Jenner and Cunningham (1944) recorded the poisoning of 62 men caused by the consumption of fruit juice prepared in a cadmium-plated vessel (see also Garber, 1946, and Monnet and Sabon 1946).

The symptoms of cadmium poisoning, which occur from 15 to 30 minutes after ingestion of the incriminated food, but occasion-

ally may be delayed as long as 4 to 5 hours, are: increased salivation, headache, vomiting, persistent diarrhoea, abdominal cramps, and acute gastritis. Later, the liver and kidneys are affected (Prodan, 1932.) The ingestion of 10 mg. ($\frac{1}{6}$ gr.) of the metal may cause marked symptoms.

Copper

This metal is normally present in most of our food animals, fruit, vegetables, and sea-foods.

Acute poisoning from the presence of copper salts in food seems to have been but rarely demonstrated, and there is no doubt that the toxicity of the metal has at times been much exaggerated. It appears to be difficult to detect ill-effects when copper is fed to experimental animals, and rats fed over a long period with food containing salts of the metals show no sign of chronic poisoning. The opinion has been put forward, however, that such experiments do not do away with the possibility of the metal having an irritating effect on certain organs of the human subject, as is known to be produced by retention of other heavy metals in the body. The salts of copper are very astringent. When the metal is absorbed in the blood in large quantities it acts as a haemolytic agent. Moreover, experiments have proved that vitamin C is destroyed by copper salts. See Schmidt-Nielsen (1936), Stotz (1937), and Mapson (1941). From some feeding experiments on healthy men copper was found to be retained in the liver, and it is inferred that such retention of the metal must be harmful.

Excessive amounts of copper (which is an active oxidation catalyst) in food would probably be avoided. For instance, its presence to the extent of about 2 p.p.m. in condensed milk gives it a tallowy flavour and in ordinary milk an emery taste. At one period, salts of copper were added to foods, vegetables and condiments to improve the colour, but this objectionable and perhaps harmful addition is now forbidden by the Public Health (Preservatives, etc., in Food) Regulations, 1925. It is, however, still practised on the Continent, and in 1934 four samples of canned mixed vegetables imported from Belgium were found to contain 36 to 54 parts of copper per million.

Investigations regarding the effects of coppered vegetables on human and animal health were made by Tschirch (1893) and Taylor, Long, and Chittenden (1913). They found no evidence of any toxic action or adverse effect on health.

For some years copper has been found present in imported concentrated tomato pulp (puree and paste). The main source of copper was found to be

Traces of copper, normally present in the tomato.

Copper salts deposited on the tomato as a result of spray during growth containing the metal. It would appear, however, from the results of investigations, that in spraying tomatoes with a copper insecticide, the spray residue does not penetrate the pulp to any extent.

Copper derived from the copper vessels in which the pulp is concentrated.

In the Food Standards (Tomato Ketchup) Order (S.J. 1949, No. 1817), amended by Regulations (S.J. 1956, No. 1167) in para. 2 of the Schedule, a maximum copper content of 20 p.p.m. in the whole of tomato ketchup, catsup sauce, and relish, on the dried solids of such products is laid down.

The amount of copper and other metals occurring naturally in foods is adequate to maintain the ionic equilibrium of these elements necessary for their proper biological functioning. Clayton (1933) compiled the following table, showing the natural copper content of foods in parts per million:

Eggs	1·1–1·19	Beef liver	16·0
White potatoes	6·5	Beef kidney	2·4
Yellow ,,	4·1	Beef tongue	1·2
Black grapes	8·1	Cow milk	0·5–0·85
Navy beans	10·45	Human ,,	0·5–0·6
Lima ,,	8·6	Butter	0·6
Yellow corn	16·6	Cheese	0·6

McCance and Widdowson (1940) also estimated and tabulated the amount of copper in various foods.

Grendel (1930) estimated that the daily diet of a child of 6 to 12 years old would contain 0·5–1·0 mg. of copper and of an adult, 2·10 mg. Thompsett (1934) calculated the average daily intake of copper to be 2 to 2·5 mg. and Leverton and Binkley (1944) 2·65 mg. The figures doubtless would vary with diet and locality.

Sea water contains about 0·01 p.p.m. of copper. The presence of copper salts in oysters is well known (and varies from a trace to several hundred parts per million) especially those obtained from the Cornish beds, but a considerable number would have to be consumed to yield a poisonous dose of the metal. Copper, if kept highly polished and free from oxide, is not readily attacked by foodstuffs. In certain foods copper appears to dissolve more readily than other metals (Tranent, 1935). A few cases of poisoning,

supposed to have been caused by food cooked in copper utensils, have been reported from time to time. Gardner (1925) mentioned an outbreak of poisoning caused by copper salts in bread which became contaminated by the copper machinery used in baking. Hebblethwaite (1928) described an outbreak of illness which was traced to the use of large copper kettles for heating water used in the preparation of foodstuffs.

The following outbreak of food poisoning recorded by Dickson (1944) is of interest:

Approximately 42 men of military units reported sick on the mornings of 11th and 12th October, complaining of violent diarrhoea, considerable abdominal pain and tenesmus which had started, awakening them from sleep, about 4 to 5 a.m. Only one patient had vomiting at the same time as the diarrhoea and none had any vomiting the previous evening. Nearly all the men affected had taken a meal at a café between 8 and 10 p.m. the previous evening, and had gone back to camp a few miles away without noting any ill-effects. A few men had lunch about 12 noon on the 10th, and they were seized with violent diarrhoea about 8 to 10 p.m. the same evening. Assuming the consumption of food at this café was the cause, the incubation period appeared to be about 8 hours. A pure enteritis developed which cleared up usually within 6 to 8 hours, even in the severest case within 48 hours. A few men in the same camp who had not visited this café also complained of slight diarrhoea, but this was atypical and most likely of a 'sympathetic' nature. No reports were received that any civilians were affected, but from the standard of notification in general, and food poisoning in particular, in this area, this is not surprising. The proprietor's wife had a typical attack on the 10th subsequent to the midday meal, although the proprietor himself did not. This can possibly be explained later.

Upon investigation the only article of food consumed by all at the café was dried peas prepared by soaking and boiling. During the week the quantity of peas used is moderate; they are cooked in saucepans of orthodox materials. On Saturdays, however, owing to the larger quantities used (particularly due to the absence of potatoes at the time) the peas were boiled in a domestic type, 15-gallon copper, made of copper and tinned by a local plumber using a proprietary tinning compound containing both flux and metal. This way had now been in use on four previous Saturdays without causing any trouble, but during the middle of the week ending the 10th a new batch of peas was used and cooked in the saucepans quite normally. On the fifth Saturday (10th October) the peas were boiled in the copper in the usual manner, but the tinning turned black up to the level of the liquid. These peas were used for the Saturday midday, evening, and Sunday evening meals, after which meals all the cases occurred A few of the peas used on the Friday (saucepan. cooked) were used up at the Saturday midday meal and might explain why the proprietor escaped although his wife was affected. This suggested fairly conclusive evidence of the toxic effect of the peas cooked in the copper. Unfortunately no peas from this cooking were available,

and the black coating on the tin lining had been scoured clean. A trial cooking was made using the same peas, soda, salt and technique; and specimens of these tinning compound, carbonate of soda used, salt, dried peas, liquid in which the peas were soaked for 12 hours, washings with soap, soda and hot water after these were removed from the copper were sent for analysis. A request was made for all samples to be analysed for copper, lead, antimony, tin and arsenic. Unfortunately the analyst saw fit to only investigate the peas from the trial cooking and the 'washing water' and these for tin and copper.

The results are as follows:

1. Peas from trial cooking . .	Tin, nil.	Copper, 10·5 gr. lb.
2. Washing water from copper after use	Tin, 2·3 gr. lb.	Copper, 22·4 gr. lb.

From the evidence available, tin and copper were present in the trial sample and possibly in greater quantity in the original cooking. The point of interest is, do tin or copper produce a pure enteritis 8 hours after consumption? All the literature I have been able to consult suggests that copper in the possible ingested dose of 5 gr. or more would give vomiting, and tin would act in a like manner. It seems most unusual that if tin and copper were present in toxic quantities that vomiting did not occur within 15 to 30 minutes. If the toxic action on the stomach was prevented by these metals being in some chemical form, possibly sulphide of lower toxicity, and only changed into a toxic salt further down in the alimentary tract, something presumably must have been in the peas to account for this. The possibility of bacterial enteritis is not overlooked, and eight rectal swabs were taken on the 12th from cases within a few hours of commencing diarrhoea; these were cultured within 1 hour of being taken, but all were negative.

Further evidence that bacterial enteritis was not the cause was that no secondary cases occurred subsequently in the camp, even amongst men sharing the same hut.

The possibility of bacterial toxin only cannot be ruled out entirely but it would have had to have been formed in the 12 hours soaking at room temperature previous to the peas being transferred to the copper and boiled and would therefore had to have been heat stable, as on the 10th they were eaten at the midday meal immediately after cooking, so that development of toxin in the cooked peas was highly improbable.

Conclusion. An outbreak of food poisoning occurred characterised by an incubation period of 6 to 8 hours—absence of vomiting and all symptoms suggesting a pure enteritis. No general symptoms other than those associated with considerable purgation, rapid recovering and complete absence of secondary cases. No evidence of bacterial infection. The noxious agent would appear to be copper dissolved from the cooking receptacle, the tin lining of which had failed. In view of a possible dose of 10 grains copper per lb. and an average portion of ½ lb. peas, the absence of vomiting is unusual, and it is suggested that some copper compound must have been formed which was inert in the stomach, but an active irritant in the intestines.

Glover (1950) mentions an outbreak of poisoning by copper which occurred among school children at Poole, Dorset, caused by

the consumption of iced lollies. These had been frozen in tinned-copper moulds. On examination of the latter it was found that the tinning had worn off in places leaving the copper exposed. The results of the analysis of a batch of the lollies which remained over showed it to contain 0·32 gr. of copper (expressed as copper sulphate) and 0·69 gr. of tin, which when expressed in gr. per lb. is 6·37 gr. of copper sulphate and 12·60 gr. of tin. The metallic contamination was assisted by the acidic watery solution of orange squash used in the preparation of the lollies.

Griffin (1951) records a case of copper poisoning caused by the consumption of coffee made with water from a gas-heated geyser.

Skone (1956) describes an outbreak of diarrhoea and vomiting following a meal consisting of boiled beef, carrots and potatoes, apple tart and custard, prepared in a school canteen at West Bromwich and served to the occupants in three schools. All the food was prepared on the same day as it was eaten. Sixty persons were affected and also 3 members of the kitchen staff. The incubation period was approximately 8 to 15 hours and the main symptoms were diarrhoea and abdominal pain. Some of the patients, however, also complained of nausea and vomiting but none were seriously ill. Specimens of faeces from the patients and samples of the meal were submitted to bacteriological examination with negative results. Nose and throat swabs taken from the kitchen staff were also negative. No chemical analysis was made. The steam-jacketed boiler used in the canteen for the preparation of the food was examined and it was found that the base plate and sides of the boiler were 'bare of tin' and there was flaking of metal on the surface of the base plate. The conditions under which the beef was prepared were recreated and afterwards portions of the cooked meat and gravy submitted for chemical analysis. The results showed that there was a varying degree of contamination with copper in different parts of the meat, ranging from 28·70 to 80·80 p.p.m. and in the gravy 1·10 p.p.m.

The symptoms of acute poisoning by salts of copper, which usually occur within 5 to 10 minutes after consumption of the contaminated food are as follows: Astringent taste with thirst, constriction in the throat, abdominal pain, diarrhoea, and sometimes collapse. In chronic cases of poisoning there is a metallic taste in the mouth, loss of appetite, dyspepsia, abdominal pain, cramps, attacks of vomiting and diarrhoea, and sometimes peripheral neuritis. A green line on the margin of the gums has been described by some observers.

The tendency in modern food factories and other places where food is prepared is to replace copper equipment by other metals such as nickel, monel, and stainless steel, and even silver, particularly as copper is readily dissolved by acid or salted foods.

Copper sulphate has been used on occasions for preventing the growth of objectionable algae in water supplies. Fowler (1905) stated that 2 pints of such water would contain $\frac{1}{4}$ gr. copper. The dose of copper as an atringent is $\frac{1}{2}$ to 2 gr. He believed that such water was harmless to consumers. Copper pipes are now in common use for the conveyance of water supplies in buildings.

The revised recommendations of the Food Standards Committee, Jan. 1956, for the limits of copper content of foods are given below:

For beverages ready-to-drink a limit of 2 p.p.m. copper, including tea. Other foods a general limit of copper of 20 p.p.m. but special limits are recommended for the present for the following foods and beverages:

Articles of Food	*Limit recommended in parts per million by weight*
(I) BEVERAGES:	
Wines, beer, cider, non-alcoholic beverages prepared from cider, and concentrated soft drinks (not including concentrates used in the manufacture of soft drinks)	7 p.p.m.
(II) OTHER FOODS:	
Chicory—dried or roasted	30 p.p.m.
Cocoa powder	70 p.p.m. on the *fat-free* substance
Coffee beans	30 p.p.m.
Colourings	30 p.p.m. on the dry *colouring matter*
Flavourings	30 p.p.m.
Edible gelatin	30 p.p.m. (already prescribed)
Pectin	30 p.p.m.
Pectin—solid	300 p.p.m.
Tea	150 p.p.m.
Tomato ketchup	50 p.p.m. on the dried *total* solids (already prescribed)
Tomato puree, paste, powder, juice and cocktails	100 p.p.m. on the dried *tomato* solids
Yeast and yeast products	60 p.p.m. on the dry matter

Limit recommended for concentrates used in manufacture of soft drinks is 20 p.p.m. No proposed special limits for shell-fish and crustacea, offals, etc. The sale of such articles containing copper in excess of 20 p.p.m. permitted if shown that the copper is of natural occurrence.

Lead

This is a familiar, dangerous, and accumulative poison, and when taken into the human body in very small quantities over a long period of time, causes chronic illness which may terminate fatally. The metal, even in the form of its most insoluble compounds, such as the sulphate or carbonate, is affected by the gastric juices and may become absorbed into the system. The chromate and arsenate are the most poisonous salts of lead. Incidentally, women are more susceptible than men to lead poisoning.

Poisoning by lead in food and beverages is mentioned in the works of Pliny, Hippocrates, and many other philosophers and writers. In the sixteenth century, Eathius describes a type of colic which was associated with the drinking of certain wines. In 1757–67 it was discovered that such wines and ciders acted upon and dissolved the glazes of the earthen vessels in which they were stored, the glaze of the vessels being compounded with lead oxide.

Beritic and Djuric (1956) describe 4 cases of lead poisoning with 1 death due to lead-glazed pottery, which is commonly used in Yugoslavia. In all 4 cases the lead was derived from wine which was heated and stored in lead-glazed jugs. Wine heated for 4 hours in vessels from one of the affected households and examined the following day contained 1·372–5·629 mg. lead per 100 c.c.

The possible effect of food or drink on the absorption of lead from the alimentary tract is a most important point and cannot be too strongly emphasized. Lead may be more rapidly and completely absorbed from liquids than from solids, which is dangerous and poisonous in the former, though not so in the latter. Experiments have established that milk interferes with the absorption of lead and is one of the antidotes prescribed for acute lead poisoning.

Every person absorbs minute quantities of lead, either through the alimentary tract from food or through the lungs from dust. It is accumulated in the teeth and bones of apparently healthy individuals in a comparatively innocuous form. Gusserow (1861) attributed this to the formation of a double salt of lead and calcium.

Minot and Aub (1924), using Fairhall's method of analysis, showed quantitatively that in animals and man nearly all the lead is stored in the bones. Fairhall studied such bones and the results of his experiments suggest that the lead is present as the very insoluble tertiary phosphate. The lead is apparently stored in the calcareous portion of the bone and not in the marrow.

In 1932 Aub, Robb, and Rossmeisl showed that lead is stored in higher concentration in the trabeculae than in the corticalis of bone.

Roche, Lynch, Slater, and Osler (1934) found from 15 to 146 parts per million of lead in various bones from healthy subjects. The bones of individuals may contain 50 to 100 p.p.m. of the metal—equivalent to 0·75 to 1·5 gm. in the whole skeleton.

A normal person may excrete daily 0·05 mg. of lead in the urine and 0·3 to 0·4 mg. in the faeces; the amount varies considerably in different individuals. Certain observers, however, are of opinion that the greater part of the metal ingested with food passes out unabsorbed in the faeces; while others contend that the intestinal canal, in addition to passing through unabsorbed lead, acts probably as the most important agent for the excretion of absorbed lead.

Cholak and Bambach (1943) found that in 1,052 normal persons with no occupational lead hazard, the mean lead concentrations were 0·030 mg. per 100 gm. of blood, 0·027 mg. per litre of urine, and 0·88 mg. per 24 hours' sample of faeces.

During the last few years investigations have proved that lead is more widely distributed in food than is appreciated, although the amount present in most foods is usually small.

Analyses have shown that lead has been found in natural foods, such as fruits, vegetables, cereals, and marine crustaceans, and that in certain foodstuffs it is sometimes present in appreciable amounts.

The British Pharmacopoeia (1932) gives the dose of lead acetate as ½ to 2 gr. equivalent to 0·3 to 1·1 gr. of the metal.

A large number of different foodstuffs were examined in the laboratory of the Ministry of Health (Monier-Williams, 1938). A considerable proportion of these contained no lead or less than 0·2 to 0·4 p.p.m., but some were found to contain lead in excess of 2 p.p.m. The following is a selection from the list of articles so examined:

Foodstuffs	*Lead in p.p.m.*
Peaches	0·9
Strawberries	0·4
Oranges (pulp)	0·5
Apples	0·3
Home-grown tomatoes	0·4
Canned peas (home-grown)	0·8
Green peas (fresh)	0·2
Rice	0·4
Self-raising flour	2·4
Milk chocolate	1·2
Sardine paste	8·3
Silds (in aluminium container)	5·1

Foodstuffs	*Lead in p.p.m.*
Bloater paste	0·9
Meat extract cubes	2·4
Baking powder (alum and phosphate)	7·1
Indian tea (loose)	10·2
China tea (in lead foil)	6·1
Custard powder	1·2
Margarine	0·3
Blancmange powder	1·0

Several relevant matters of importance in connection with the presence of lead in food require special consideration. Until recently the determination of very small quantities in food was extremely difficult, and methods for analysis could not be relied upon to give uniformly accurate results, especially when only minute traces of the metal were present. Monier-Williams (1938) found that of the many possible combinations of published methods is one which includes extraction with 'dithizone' and precipitation of the lead as sulphate and determination colorimetrically as sulphide. This method is capable of determining minute amounts (0·002 mg.) of lead.

Of late years considerable controversy has arisen regarding the amount of lead in food which may be considered negligible from a health point of view.

It has been calculated that 2 mg. of lead ($\frac{1}{32}$ gr.) absorbed daily, undermines the constitution and may set up chronic poisoning with changes in the kidneys and arteries which shorten life. A daily intake of 1 mg. or even less must be regarded with suspicion.

Analyses of the food examined in the laboratory of the Ministry of Health, reveal that normally about 0·2 to 0·25 mg. of lead is likely to be ingested daily with food and that the total intake from all sources would be 0·5 mg. of the metal (1·22 mg. in food, 0·20 mg. in water, and 0·08 mg. inhaled as dust). If, however, certain items are added to the diet the total amount of lead ingested would become excessive. Ingleson (1938) showed that the average content of lead in water supplies was 0·118 p.p.m.

While the lead content in the majority of foods, is very small, and its further reduction may be impossible, the metal may be present in some foods in considerable or even excessive amounts. The question arises whether it would be possible by the introduction of a standard (by specific legislation) either to eliminate the lead content in foods or reduce the amount to safe limits.

Monier-Williams (1938, 'Lead in Food') remarks:

The presence of lead in any particular food must be regarded, not only as a danger in itself, but as a contribution, more or less serious, to

the total daily intake of lead from all sources. The aim should be to reduce the total amount of lead ingested in the diet to the lowest possible amount, and to this end every endeavour should be made to ensure that individual foods are prepared in such a way as to eliminate lead contamination as far as possible.

The Chief Medical Officer of the Ministry of Health (1938) in the Prefatory Note to the above pamphlet, sums up the matter as follows:

We are at present unable to say what quantity of lead may be considered negligible in food. It is, however, reasonable to infer that the harmful effects of continued small doses of lead begin from the moment the lead is absorbed and that the crude symptom-complex of chronic poisoning is merely the final stage of a long series of more subtle metabolic disturbances which elude our imperfect methods of detection. In other words, the obviously harmful effect on the normal activities of the body of continued small doses of lead would seem to justify the assumption that there is no threshold below which still smaller doses can be regarded as being without some adverse effect. It would appear therefore that complete absence of lead from food is the ideal to be aimed at. For this reason it would seem inadvisable to set up standards by specific legislation which, by fixing permissible limits of contamination, would inevitably impede efforts to secure the reduction of lead in food to the lowest possible amounts. Our object must be to reduce the amount of any toxic substance in food to the smallest that can be achieved in practice, and this in many cases may be attained more effectively by administrative action than by the prescription of specific standards.

Food may be contaminated by lead in five sorts of ways:

One way is exposure of food to dust containing lead as produced by the disintegration of lead pigments and paints during weathering.

A second source is the utilization of solders, alloys, enamels, and glazes containing lead, in the construction of receptacles, plant, machinery, and apparatus, etc., which may come into close contact with food and food products, including certain beverages.

In a report to the Ministry of Health (Monier-Williams, 1925) on the 'Solubility of Glazes and Enamels in Cooking Utensils', it was shown that food cooked in utensils having lead glazes might take up 3 to 4 parts of lead per million. This amount is increased if the food is allowed to remain in the vessels for any considerable length of time. He states:

The probability that undesirable constituents in insignificant amounts may be dissolved from enamelled hollow-ware during the ordinary processes of cooking may be regarded as remote.

Savage (1920) quotes an interesting case observed by Halenke and reported to Lehmann (1902). Two women ate cranberry tart for which they had cooked the cranberries in a cheap earthenware pot. Soon after eating part of the tart they became ill, one severely so. The glaze had been dissolved from inside the pot. A piece of the tart contained 160 mg. of lead. It was estimated that each woman had consumed from 400 to 600 mg. of malate of lead and that approximately as much as 100 mg. had been dissolved in this single cooking. Cooking utensils now manufactured in Great Britain have a leadless glaze.

The metal is attacked by acids, alkaline foods, and beverages, and many instances of poisoning have been recorded as a result of such contamination both in this country and abroad.

Beer and cider are known to take up lead from vessels and pipes. In 1922, 93 cases with 1 death occurred in the County of Middlesex from the consumption of beer which had dissolved a substantial proportion of lead (up to 1·9 gr. per gallon) from the enamel linings of the tanks in which it was stored.

Chronic lead poisoning occurred in a metropolitan borough in 1936. The beer had been drawn from the barrels through old lead piping. On analysis it showed the presence of 1 p.p.m. of lead.

Jackson and Jackson (1932) reported an outbreak of lead colic from Devonshire. This was traced to cider drawn through tin-washed lead pipes connected to the casks and counter engines. Lead was present to the extent of $\frac{1}{10}$ to $\frac{1}{20}$ gr. per gallon.

Samples of beer recently examined by local authorities showed from 0·3 to 3·0 and occasionally 9 and 13 p.p.m. of lead.

Bodron (1925) reported a curious epidemic of 37 cases of lead poisoning. This was traced to bread baked in an oven heated by wood derived from the breaking up of old boats, the wood being impregnated with paint containing lead salts. The vapour condensed on the loaves in the oven.

In modern public houses and hotels blocked tin pipes have taken the place of lead pipes. In many cases the lead pipes have been tin-washed, which affords little protection from the corrosive action of cider. Lead pipes lined with tin are not reliable unless the tin lining is thick and not damaged or worn. It has been suggested that pipes made of selected corrosion-resisting steel alloy for use with beer might be satisfactory.

The use of tin-washed and tin-lined pipes for beer and cider was discussed in the Annual Reports of the Chief Medical Officer to the Ministry of Health for the years 1932 and 1936 respectively.

The tin coating of tin-plate may contain small amounts of lead (less than 0·1 per cent or even more in the case of commercial block tins), and it is possible that traces of lead find their way into the food.

In the Annual Report of the Chief Medical Officer of the Ministry of Health for 1935, attention is drawn to the importance of tea imported in lead-lined chests. Analyses of samples from these chests indicate that dry tea may contain considerable amounts of lead dust—varying from 10 to 20 p.p.m. Experiments have shown that about one-third of this lead goes into solution or suspension in the tea infusion as consumed. About 2 p.p.m. of lead in tea seems to be unavoidable as it becomes contaminated during the processes to which the leaf is subjected. The amount of lead in tea can be greatly reduced by using stout paper liners. Aluminium has been used with success in place of lead-lined chests.

In the United States the limit of lead in tea is 2·50 p.p.m.

The Food Standards (Curry powder) S.J. Order 1949 No. 1816 (Amended by S.J. 1956, No. 1166) says that no curry powder shall contain lead in excess of 20 parts of lead per million parts of curry powder.

Lead may gain access in small quantities to foil-wrapped articles, such as cheese and confectionery, but the use of paper interliners prevents contact between the food and the foil.

Trouble has been experienced with imported sardines which were found to be contaminated with lead. Consignments representing thousands of tins of these fish were rejected at the Port of London. The subject was discussed in the Annual Report of the Chief Medical Officer of the Ministry of Health for 1936. In 50 samples examined by local authorities the lead content ranged from 10 to 80 p.p.m.

Lampitt and Rooks (1933) gave the results of the examination of 596 samples, 30 per cent of which contained from 10 to 90 parts of lead per million.

The contamination of the fish was derived from the grills (iron wire coated with solder containing a high proportion of lead) on which they were steamed. Steam condensing on the grills takes up the lead and contaminates the sardines.

At a Conference of Port Medical Officers of Health in 1933 it was agreed that sardines should be free from lead or contain negligible traces of the metal. As this would necessitate alterations to plant, etc., it was decided provisionally to take no action in cases where the lead content did not exceed 20 p.p.m. As a result, a

marked improvement took place, and at a second conference in 1937 the provisional limit was reduced to 5 p.p.m. for a limited period. Ultimately sardines will be required to be free from lead or contain only negligible traces.

Lead has been present in smaller quantities in a number of other canned products, for example, tunny fish 13 p.p.m. *pâté de foie gras* 10 p.p.m., anchovies 8 p.p.m., peeled shrimps 7 p.p.m., crab paste 6 p.p.m.

The Medical Officer of Health for the Port of London in his annual report for 1938 remarks: 'Merchants have argued that the amount of lead in sardines is not dangerous to health, and have told me how many tins of sardines they have eaten, but when it is pointed out to them that lead is an accumulative poison, that much damage may be done before definite symptoms of poisoning can be diagnosed, and that the trouble is not just the quantity ingested in sardines but the many small doses from the many different sources, they see our point of view and are anxious to know what steps can be taken to eliminate lead from their products.'

A third source of lead is the use of citric and tartaric acids, cream of tartar and acid calcium phosphate, synthetic dyes, etc., in the production of which materials containing lead have been used.

The contamination of citric and tartaric acids and acid calcium phosphate is mainly due to the use of lead utensils for concentrating and crystallizing these chemicals. In 1907 (Local Government Reports of Inspector of Foods No. 2), at a special inquiry, the conclusion was reached that amounts of lead not exceeding 20 p.p.m. would not be considered sufficient to justify their condemnation.

The British Pharmacopoeia gives the limit of lead in the above chemicals as 20 p.p.m. There have been, however, vast improvements in the production of the above articles, and the acids can now be obtained with lead content of less than 2 p.p.m.

With regard to the use of artificial colouring substances the Public Health (Preservatives, etc., in Food) Regulations of 1925 prohibit the use of metallic colouring matters and compounds of lead for colouring food.

Food colouring materials are used in small amounts in many food products. The colours are carefully prepared for these purposes and standardized as regards tinctorial power; they generally contain traces only of arsenic and deleterious metals such as lead and copper. In the main it may be stated that colours

for foodstuffs contain less than 5 parts of arsenic per million and less than 50 parts of lead per million, while many fall below these limits.

A fourth source of food contamination is the spraying of fruits and vegetables with insecticides containing lead compounds.

From time to time lead arsenate and other lead compounds have been found in excessive amounts in the wrappings and skins of imported apples and pears. In one case, $\frac{2}{3}$ gr. per lb. was present in the wrappings and $\frac{1}{12}$ gr. per lb. in the skins. These chemicals are widely used as insecticidal sprays on fruits and vegetables and mixed with substances to prevent them being washed off by rain. It was found at first, however, that brushing and wiping would not remove the poisonous residues, but later improved methods were adopted for cleaning the fruit before shipment, which have been effective in reducing the lead content to minute proportions.

The following is an extract from a circular issued in 1935 by the United States Department of Agricultural Food and Drug Administration, Washington, DC.:

> Lead Arsenate Sprays—While lead arsenate sprays are no longer necessary for controlling the insect pests of vegetables, there is as yet no less toxic substitute in the production of apples and pears. However, the commercial cleaning of fruit has become practically universal, and has been perfected to the point where the intake of poison from this source is very much less than at any time in recent years. With continued Federal and increasing local vigilance, the danger to health will never again become significant. We have advised consumers that if they wish to make assurance doubly sure, they may remove any last vestages of poison spray that may be present by cutting out the natural 'cups' of the fruit at stem and blossom ends and discarding the peel.

The fifth case is the presence of lead in shell-fish and crustacea.

Chapman (1926) made investigations into the presence of lead in shell-fish and crustaceans. He was of opinion that the lead was derived from the sea water.

Shell-fish and crustaceans examined in the laboratory of the Ministry of Health yielded the following results:

	Lead in p.p.m.		*Lead in p.p.m.*
Oysters	0·2	Crab	0·3
Lobster, shell	3·4	Winkles	1·5
Whelks, A	0·7	Shrimps (in aluminium container)	0·3
Whelks, B	2·1		

The Edible Gelatin (Control) Order, 1947 prescribes a limit of 10 p.p.m. of lead in gelatin.

FOOD STANDARDS COMMITTEE: RECOMMENDATIONS FOR LIMITS FOR LEAD IN FOODS (1954)

Article of Food	*Limit recommended in parts per million by weight*
(I) BEVERAGES	
Concentrated soft drinks (but not including concentrates used in the manufacture of soft drinks)	0·5 p.p.m.
Brandy, gin and geneva, rum and whisky	
Wines, liqueurs, alcoholic cordials and cocktails and alcoholic liquors not otherwise specified	
Beer	
Cider	1 p.p.m.
Fruit and vegetable juices (including tomato juice but not including lime juice and lemon juice)	
(II) OTHER FOODS	
Anhydrous dextrose and dextrose monohydrate	
Edible oils and fats	0·5 p.p.m.
Refined white sugar (sulphated ash content not exceeding 0·03 per cent)	
Ice cream, iced lollies and similar frozen confections	1 p.p.m.
Canned fish	
Canned meats	
Dried or dehydrated vegetables (other than onions	
Tomato puree paste or powder containing 25 per cent or more total solids	
Edible gelatin	5 p.p.m.
Meat extracts and hydrolysed protein	
All types of sugar, sugar syrups, invert sugar and direct consumption coloured sugars with a sulphated ash content exceeding 1·0 per cent	
Raw sugars except those sold for direct consumption or used for manufacturing purposes other than the manufacture of refined sugar	
Edible molasses	5 p.p.m.
Caramel	
Liquid and solid glucose and starch conversion products with a sulphated ash content exceeding 1·0 per cent	5 p.p.m.
Cocoa powder	5 p.p.m. on the dry fat-free substance
Yeast and yeast products	5 p.p.m. on the dry matter
Chemicals which may be used in foods and for which a lead limit is specified in the B.P. or B.P.C.	The limits specified in the edition of the B.P. or B.P.C. current from time to time
Tea	
Dehydrated onions	
Dried herbs and spices	
Flavourings	
Alginic acid, alginates, agar, carrageen and similar products derived from sea-weed	10 p.p.m.
Liquid pectin	
Chemicals not otherwise specified, used as ingredients or in the preparation or processing of foods	
Food colourings (other than caramel)	20 p.p.m. on the dry *colouring* matter
Solid pectin	50 p.p.m.

Lolly ices can be contaminated by metals. Owing to the ever growing popularity of lolly ices among children of all ages throughout the whole country, Semple (1953) carried out some observations in Liverpool on the incidence of metallic contamination of these articles. Of 70 samples examined, 39 were found contaminated; 6 contained lead, 14 copper, 6 tin, and others more than one metal. Semple remarks:

> It does appear that the various contaminating metals were probably introduced from the moulds. The main source of lead was probably from tin plate of poor quality containing lead or from moulds soldered with lead solder, whilst the copper and zinc found in the samples resulted from worn and scratched moulds. It might be considered that some standard of composition is advisable in view of the low nutritive value of the average lolly ice.

So far as shell-fish and crustacea in which lead may occur naturally in amounts higher than 2 p.p.m. are concerned, the Sub-Committee recommended that the sale of these foods containing lead in excess of 2 p.p.m. be permitted if it can be shown that the lead is natural to the fish.

Aluminium

Probably no metal has caused so much controversy as the question of the toxicity of aluminium and its salts. While the extensive investigations and experiments carried out from time to time by numerous observers to ascertain the amount of contamination of foods cooked in aluminium vessels and their effect on the human system have frequently given negative results, statements continue to appear questioning the wholesomeness of repeated ingestion of this metal and its salts. The issue is raised again and again by those opposed to its use, and consequently the literature on the subject has become voluminous.

Plagge and Lebbin (1893) conducted experiments in their laboratory. For 18 months the midday meal, which consisted of coffee and vegetables, was prepared and cooked for 2 men in aluminium vessels. No metallic taste was noticed, the vessels proving satisfactory. The 2 men put on weight and remained in good health. The observers concluded that aluminium plates are attacked by most foods, but the amount of the metal taken by a person in a day is only a few milligrams.

In 1913 *The Lancet* published the results of investigations upon the effects of cooking foods in aluminium vessels. The experiments were carried out under conditions similar to those found in ordinary kitchens. Various foods and beverages were cooked in aluminium

vessels and the amount of the metal found in the food estimated and the effect upon the utensils studied. The report concluded:

> We are confident that aluminium, as it is now made by reputable manufacturers, is a suitable material for cooking vessels, and that any suspicion that it may communicate poisonous qualities to food in the process of cooking may safely be dismissed in view of the results of the practical experiments which we have recorded, showing that the metal is not appreciably acted upon in cooking operations. This finding is satisfactory also, inasmuch as aluminium is an excellent heat conductor; cooking in aluminium vessels is, therefore, rapid, and fuel is economised in consequence. But the management of aluminium cooking utensils requires the same ordinary applications of common sense as are customary in case of other metals employed for a similar purpose.

Thieme (1929) demonstrated by tests on experimental animals the suitability of pure aluminium vessels for culinary purposes. His experiments extended over several months, and he concluded that abnormally large doses of aluminium salts are devoid of deleterious physiological action.

The conclusions after six years' investigation at the Mellon Institute, Pennsylvania, U.S.A. (1933) are: (1) aluminium is not a poisonous metal and does not give rise to any disease, (2) aluminium vessels are very resistant to corrosion by foodstuffs cooked therein, and (3) aluminium does not accelerate the destruction of vitamins or other food accessory substances during cooking.

Monier-Williams (1935) in a special report to the Ministry of Health on 'Aluminium in Food' arrived at the following conclusions:

> Much of the experimental work which has been carried out to ascertain whether aluminium in food is harmful or not is conflicting and inconclusive. Aluminium salts, in doses which are not unreasonably high, have been shown to be not without action on digestive processes. It is safe rule to exclude from food as far as possible anything which may reasonably come under suspicion of causing harm, and on this account it is undesirable to admit aluminium in the relatively large amounts in which it may be employed as a constituent of baking powders or self-raising flour.
>
> There is, however, no convincing evidence that aluminium in the amounts in which it is likely to be consumed as a result of using aluminium utensils has a harmful effect upon the ordinary consumer. It is possible that there may be individuals who are susceptible to even such small doses of aluminium as may be derived from aluminium utensils, but evidence of this is inconclusive.

If ordinary London tap water is boiled in aluminium utensils and these are allowed to stand overnight, 8 to 20 p.p.m. of the metal is taken up by the water.

Wührer (1939) reports that evidence, gathered from experiences in various countries, justifies the conclusion that hygienic objections no longer exist to the use of aluminium in contact with raw and cooked foods.

With reference to the addition of alum to flour for the purpose of arresting fermentation or renovating flour damage by damp storage, this was forbidden by the Bread Acts of 1882 and 1886.

Under the Sale of Food and Drugs Act, 1875, several prosecutions took place as a result of using sodium aluminium sulphate—commercial baking powder—on the ground that it caused gastric troubles. As a result phosphate powders took the place of alum baking powders, but attempts have been made to revive the trade of alum baking powders. Investigations on experimental animals, however, have shown that such baking powder interferes with growth and with the reproductive functions.

Because of its lightness and durability, the use of aluminium has increased considerably during recent years in the construction of household utensils, dairy equipment, brewery plant, containers for milk, preserved meat, vegetables, fish and shell-fish. The metal foil is also utilized for wrapping articles of food. The fact that the specific gravity of aluminium is about one-third of tinned steel is a distinct advantage in handling, transport, and shipment. The metal, ordinarily, is not subject to galvanic corrosion and other disadvantages of a plated metal. Aluminium cans do not blacken. Fish packed in oil, however, sometimes develop hydrogen, resulting in the containers becoming blown, so that internal protection of the can is necessary.

Lacquering presents no difficulty and anodic oxidation gives added protection and improves the adhesion of the lacquer. In Norway, brisling and other fish products in tomato sauce are packed in anodized and lacquered aluminium cans of 99 per cent purity. Anodized (not lacquered) metal is used for most other foods, including brisling and herring in oil, shrimps, crab meat, cod's roe, meat products, beans and peas. Acid products such as fruits, etc., are packed in anodized or lacquered aluminium cans. Recommendations have been made that aluminium alloy be used for lining the holds of trawlers to protect the fish and ensure greater cleanliness.

Kaess (1948) reports on the uses of aluminium foil. Faulty manufacture may result in the presence on these foils of pores which can have unfavourable effects on the goods packed in them. Thin foils often show a highly porous structure. Different results

were obtained for varnished and covered foils. Such foils may be used with advantage for wrapping hydroscopic foods.

In 1950, the British Standards Institution published a standard for 'Coated aluminium foil for wrapping cheese', B.S. 1683. It specifies chemical composition of the uncoated foil, tolerance of thickness, limits of perforation in the metal, details of the protective coating, methods of sampling, and includes appendices giving methods of test.

In planning further research with a view to extending the usefulness of aluminium, the Low Temperature Research Station, Cambridge (Bryan, 1948) carried out an intensive survey of all the available information relating to the properties of aluminium and its alloys with special reference to corrosion and its prevention. This information is now available in a comprehensive Special Report No. 50, issued by the Department of Scientific and Industrial Research.

Tin

The wide use of tin-plate for the construction of receptacles for the preparation, storage, and canning of foodstuffs and tinfoil for the wrapping of perishable articles, makes contamination from these sources of considerable interest to food manufacturers. Chemical changes take place between fruit juices when heated in the presence of tin, and the metal is especially taken up by foods containing acids, such as meat extracts, vegetables, vegetable soups, and fruits including peaches, apricots, pears, cherries, pineapples, asparagus, and tomatoes. Adams and Horner (1937) in their researches on canned fruit and vegetables showed that in double-lacquered cans the content seldom exceeds 40 p.p.m. and in plain cans and with most foods under normal conditions it was usually below 100 p.p.m. For some reason sardines, silds, herrings, and similar fish canned in oil or tomato sauce are particularly prone to attack the surface of tin-plate, and 2 to 8 gr. to the lb. have been found present. In some cases the containers were almost completely de-tinned, the fish actually sticking to the inside of the can. It would appear that in some instances the tin content in food increases if the can is opened and the contents exposed to the air for some time. Glassmann and Barzutzkaja (1928) found that when an opened can of fish was exposed to the air for 12 days, the tin content increased from 154 to 420 p.p.m. The formation of sulphides of iron and tin from the sulphur in certain foods sometimes forms a bluish sheen or marbled appearance

on the tin. No corrosion is indicated, in fact a film of such sulphide seems to provide protection during storage against acid juices.

In this connection it is of interest to mention the 'Protecta-Tin' process which was evolved in the laboratories of the Tin Research Institute, and introduced into the canning industry for the purpose of preventing the sulphur-staining of the inside of cans by producing an invisible, resistant oxidation film on tin-plate. The film is produced by immersing the tin-plate in a hot alkaline phosphate-chromate solution containing a suitable wetting or penetrating agent, which simultaneously degreases and films the tin-plate (Kerr, 1940). The process is inexpensive, the film produced is quite harmless to food and there is no health hazard in its application.

Trials were carried out at the Fruit and Vegetable Preservation Research Station, Chipping Campden (University of Bristol) and at the British Food Manufacturers' Research Association, also by several of the leading canners in this country and abroad. These extended trials showed the treatment to be most efficacious with meat, kidney soup, brawn, and meat gelatine cans.

In a subsequent investigation it was shown that the time of the filming treatment could be reduced from five minutes to about thirty seconds, by simple modifications to the make-up of the filming bath. This investigation (Kerr, 1946) also dealt with the application of the filming treatment to tin-plate sheets prior to fabrication.

Besides giving protection against internal sulphide staining, the film also greatly increases the resistance of tin-plate to ordinary atmospheric rusting. This is a point of considerable importance when cans may be stored in damp atmospheres or be subjected to atmospheres below the dew-point during transport. It will be observed that the treatment of the tin-plate prior to fabrication enables these two advantages to be combined, since the container is by one operation made proof against sulphide staining and atmospheric rusting.

A further advantage is that in a range of experimental packs the amount of absorption of tin by the food during sterilization and storage was found to be less in filmed cans than in unfilmed control cans. On the whole, the tin content of the food in the treated cans was from 30 to 40 per cent less than in the control cans.

The present state of the development of the process is described in a booklet published by the Tin Research Institute, Fraser Road, Greenford, Middlesex (1950) and also in a pamphlet on working instructions for the 'Protecta-Tin' treatment.

A considerable difference of opinion seems to exist regarding the toxicity of tin. Apparently there is no reliable data that the metal is harmful. Most investigations point to the view that tin does not ordinarily affect the human system. Schryver (1908) concluded from all the different investigations that 'they do not indicate much probability of serious risk of chronic poisoning by the absorption of non-irritant compounds of tin as a result of diet which consists largely of canned foods and is continued over considerable periods of time.'

Animal experiments and investigations have been carried out by many observers including Lehmann (1902), Eckardt (1909), Eber (1910), Salant, Rieger, and Treuthardt (1914), Goss (1917), Salant (1920), Flinn and Inouye (1928). The results showed that fairly large doses are needed to cause fatal results in animals. About 4 to 6 gr. of tin chloride are required to kill a dog.

In the view of Buchanan and Schryver (1908), 'it seems clear that, in any kind of canned food, quantities of tin approximating to 2 gr. to the lb., are not only unusual and unnecessary, but must also be regarded with grave suspicion in consequence of the risk of irritant action of the tin they contain.

Buchanan also drew attention to the desirability from an administrative point of view of requiring the date and place of preparation to be shown on the labels or to be otherwise available when required.

It is generally agreed that foods consumed within a few months of canning may contain as much as $\frac{1}{2}$ gr. of tin to the lb., but this does not ordinarily cause gastro-intestinal irritation in the amount usually taken at a single meal. Savage (1920) says:

> Tin may exert a toxic action in two definite ways. The amount taken into the body with the food may be so considerable that a single dose may set up acute symptoms or chronic poisoning may be induced by much smaller quantities taken over a long period.

Considerable apprehension used to exist in the minds of many concerning the presence of metals in food wrapped in foil, although this protects the food from bacteria and dirt. In 1929 the Ministry of Health drew attention to the increasing practice of wrapping foods, such as soft cheeses, confectionery, etc., in tinfoil and to the possibility that, in some cases, excessive amounts of tin may be taken up by food. It was suggested that manufacturers should give their attention to the matter with a view to substituting grease-proof paper or similar material for tinfoil.

The Annual Report of the Chief Medical Officer, Ministry of Health, 1932, states:

In Hammersmith a number of soft cheeses wrapped in tinfoil were found to be badly blackened and mouldy and were seized and destroyed. A further sample was taken under the Sale of Food and Drugs Act and was found to contain 14 grains of tin per lb. A conviction was obtained.

In 1948, under the authority of the Dairying Industry Standards Committee of the British Standards Institution, B.S. 1436 for coated tinfoil for wrapping cheese was issued.

It is well known that coated tinfoil of inferior quality will lead to the rapid deterioration of the cheese with which it has been used and the standard has, therefore, been prepared to lay down a minimum quality, although it is realized that wide difference in the hardness and ductility of coated foils may be required by cheese manufacturers.

The British Standard was drafted with a view to:

1. Establishing a limit to the quantity of hardening constituents in the metal within the reasonable demands of the trade today.

2. Limiting the total impurities permissible in the metal.

3. Establishing tests that would indicate the suitability of the mechanical properties of coated foil and the protective value of the lacquer coating.

4. Setting forth the gauges of foil required.

5. Restricting the amount and establishing the quality of coating.

6. Establishing the limits of perforation beyond which the foil may be regarded as unsatisfactory.

7. Describing methods of sampling.

8. Describing methods of analysis and test.

In 1952, The Food Standards Committee adopted a report by its Metallic Contamination Sub-Committee in respect of the limits of tin in canned foods. The suggested limit of 2 gr. of tin per lb. (286 p.p.m.) for canned foods, proposed by Buchanan and Schryver in 1908, appears to have been acceptable in practice. The Committee is of opinion that a limit of tin should be maintained in the interests of good commercial practice. It is proposed that an informal limit of 250 p.p.m. be accepted, but that no statutory effect be given to the limit. No recommendations have been made with regard to food and beverages which are not subjected to a canning process.

Acute tin poisoning is rare. The symptoms are characterized by colic, meteorism, oppression in the chest, constipation, and later by a metallic taste in the mouth.

Zinc

This metal is normally present in the liver and spleen of man and animals (Rost, 1921). It is widely distributed in various foods, such as cereals, milk, eggs, oysters, fish, vegetables, edible meats, marine animals, and in certain drinking waters. Soils may contain from 1 to 5 p.p.m. of zinc.

Severy (1923) records the average amount of zinc found in samples of different marine animals as follows:

Animal	*No. of samples*	*Average amount of zinc for samples mg. per kilo*
Sea anemone	2	10·50
Starfish (black)	3	20·72
Starfish (yellow)	2	15·70
Starfish (red)	3	19·06
Sea urchin	2	2·11
Yellow slug	6	31·00
California oyster	8	64·97
Clams	3	11·63
Mussels	4	22·45
Abalone	5	24·12
Cryptochiton	17	12·67
Shrimp	6	18·65
Crab	9	30·97
Salmon	2	8·00
Sea-lion	17	32·25
Whale	6	40·00

There is little evidence that the continued ingestion of small amounts of this metal has any deleterious effect on man, and despite the fact that outbreaks of food poisoning are now and then attributed to the contamination of foodstuffs by the metal, experiments have usually not confirmed these toxic properties.

Clayton (1933) remarks:

> The normal intake of zinc per day in food by an adult is about 12–15 mg. and the normal adult excretes zinc in the urine (0·2–52·0 mg.) and faeces (2·67–19·9 mg.). Human milk has been found to contain 3·89 p.p.m., and cow milk 4·58 p.p.m. of zinc. Analyses of Bertrand and Benzon (1928) showed zinc content in p.p.m. potatoes 5: garlic 10: onion 13·8: peas 44·5: cereals 12–19·5: lentils 24·4: polished rice 2·5.

Other observers have recorded 0·20 to 2 p.p.m. in vegetables, oranges, lemons, apples, and fruits of a soft nature.

The use of galvanized vessels in modern food factories is practically unknown, but vessels lined with zinc are sometimes used for the storage of foods. Salkowski records that a plum marmalade prepared in a vessel coated with zinc yielded 3·4 per cent of this metal calculated as sulphate. Acid foods are able to dissolve considerable amounts of the metal from galvanized vessels. Investigations made by Sale and Badger (1924) on the effects of various liquids in zinc vessels is shown in the following table, one gallon of liquid (except in the case of milk, 1 quart) having been placed in a galvanized iron pail:

	Zinc as p.p.m.	
	After 17 *hours' contact*	41 *hours' contact*
Tap water	5	21
Distilled water	9	27
Carbonated water	193	181
Milk	438	1,054
Orangeade	530	854
Lemonade	1,411	2,700

Allport and Moon (1939) found 0·23 p.p.m. of zinc in London tap water. Kerr (1942) ascertained that caps for milk bottles (composed of tin-zinc alloy) absorbed the cream layer on the milk to the extent of 50 to 60 p.p.m.

Poisoning by zinc and its salts is not unknown and occasional outbreaks have been recorded, one of which occurred in Surrey in 1922. Two hundred persons were served with apples which had been stewed in galvanized iron pans and all suffered from dizziness, vomiting, colic, and diarrhoea. The illness only lasted a few hours and all recovered. It was estimated that each person consumed zinc equal to about 20 gr. of sulphate of zinc.

Several cases of zinc poisoning were reported from Hereford in 1943, caused by the consumption of apple rings which had been cooked in galvanized iron steamers.

Within three-quarters of an hour of breakfast at an A.T.S. depôt the majority of the auxiliaries who had taken the meal in two adjacent canteens became violently sick. The vomiting was followed in many cases by diarrhoea, cramps, and a varying degree of collapse. All recovered within 24 hours.

Prompt action by the Medical Officer secured small portions of (*a*) steamed or boiled fish, and (*b*) steamed apple rings, which were sent to the laboratory. A bacteriological examination having failed to yield any suspicious finding, the possibility of metallic poisoning was explored. It was then found that a watery extract of the apple gave a strongly positive reaction for zinc, using a 'spot test' with acridine hydrochloride.

Professor Delafield kindly examined the remainder of the apple, which had now been triturated with equal parts W/V of water, and

eported zinc present in a concentration of 0·12 per cent, which expressed as crystalline sulphate equals 0·53 per cent. It was therefore evident that a helping of the apple rings weighing 4 oz. would contain about 1·0 g. of zinc in terms of the sulphate, the emetic dose of which is said to be 0·6 to 2·0 g.

Inquiry revealed that auxiliaries attached to two cook houses had been affected, and that the preparation of the apple had not been identical in both cook houses. In one, after soaking overnight in large mess-tins, the apple rings had been transferred to a skep of a galvanised iron steamer for cooking; in the other, the tins were placed in the steamer direct. It appeared probable that, in the former method of treatment, zinc would be readily taken up during the cooking, but it was difficult to account for the access of the metal in the latter.

A similar outbreak of zinc poisoning due to cooked apple rings was recorded by Tomlinson (1944). This occurred in November, 1943, at an R.A.F. establishment in East Anglia.

About 200 R.A.F. personnel suffered from nausea, vomiting and some degree of prostration 5 to 10 minutes after eating stewed apple rings for lunch. About 20 subjects had to be admitted to the station sick quarters. Only those who had eaten the apples were affected. There was no diarrhoea. The dried apple rings were cooked in a galvanised iron vessel which was said to have been used for this purpose before. The cooked apple was reported to have a sharp metallic taste.

The analysis and report were as follows:

The wet, uncooked rings contained 0·034 per cent zinc. The wet, cooked apple rings contain 0·141 per cent zinc. No lead, copper or arsenic was detected in either of the specimens. . . . Expressed as zinc sulphate, the cooked apple rings contain 0·642 per cent. A 4 oz. helping would contain about 11–12 grains of zinc sulphate. The B.P. emetic dose of zinc sulphate is 10–30 grains.

In 1946, zinc from a galvanized iron vessel in which rhubarb tart had been cooked was responsible for the sickness of 20 children who ate the tart and quickly rejected it. The metal was recovered from a similar tart cooked in the same way in the same vessel.

Outbreaks of food poisoning due to the consumption of fruit or fruit juices of various kinds which have been prepared or stored in zinc vessels have been recorded from time to time. It would therefore appear essential that the public should be warned against this dangerous practice.

The Food Standards Committee, Ministry of Food (1953) were satisfied that public health requirements would be met by observing limits of zinc consistent with efficient commercial practice as follows: Beverages ready to drink 5 p.p.m. Other foods 50 p.p.m.

For edible gelatin, however, a maximum of 100 p.p.m. is recommended. The Committee are of the opinion that

in view of the high natural zinc content of certain animal and vegetable products, e.g. herrings, shell-fish, and crustacea, cereal offals and animal offals, etc. we consider that no objection should be taken to the sale of such articles containing zinc in excess of 50 p.p.m. if it can be shown that the zinc is of natural occurrence.

Sodium Fluoride

This poisonous substance has been used extensively of late years (owing, probably, to the shortage of other suitable insecticides) either alone or mixed with other ingredients for the extermination of household pests, particularly cockroaches. The percentage of sodium fluoride in the insecticide powders varies, but the poisonous properties of the substance are still there. The powder is usually sprinkled on the floors in infested bakeries, kitchens, restaurants, canteens, etc., or is sometimes applied by means of a powder bellows to facilitate contact with the insects. Unless proper precautions are taken, the powder is liable to contaminate any food left exposed on the premises. Korff and Kaplan (1942) report:

Accidental poisoning from white sodium fluoride is not uncommon. In 1941 the Maryland State Board of Health (U.S.A.) adopted regulations requiring that insecticides containing sodium fluoride shall be coloured Nile blue as a warning. These regulations were adopted because of the widespread use of insecticides containing uncoloured sodium fluoride in hotel, restaurant, and hospital kitchens in Maryland. Inspectors are equipped with vials of fluoride test paper. The test depends on the fact that, in the presence of strong acids, soluble fluorides decolorise the zirconium lake of sodium alizarin sulfonate. Strips of filter paper are impregnated with the fluoride reagent and are then dried. The test is made by moistening a test paper with dilute hydrochloric acid (the orthotolidine solution carried by the inspectors for the residual chlorine test may be used). The moist strip is then touched to the suspected powder and, in the presence of a large amount of soluble fluoride, decolorisation of the paper will occur in about 5 seconds. Decolorisation is evident in about 30 seconds when a 1 to 1,000 dilution of sodium fluoride powder in flour is used, A reaction time of 5 minutes should be allowed for traces of fluoride. Phosphates and oxalates may yield false tests and therefore positive findings should be confirmed in the laboratory.

Sodium fluoride is a white powder which somewhat resembles such culinary articles as baking powder, bicarbonate of soda, cream of tartar, etc. Cases have been recorded from time to time where this poisonous substance has been stored on the premises and used in mistake for baking powder (Baldwin, 1899), with

serious and even fatal results. According to Carr (1936) 3 gm. of of sodium fluoride is sufficient to cause death in man.

The symptoms of fluoride poisoning occur in a very short time after the consumption of the contaminated food. This chemical is extremely irritating to the stomach mucosa, rapidly producing marked congestion and erosion in high concentration. The first symptoms are a sense of burning and constriction in mouth and throat, salivation and nausea followed by vomiting, diarrhoea, cramps, and abdominal pain; there may be convulsions and partial paralysis. Death may result from shock, respiratory or cardiac failure or asphyxia due to convulsions. In acute cases death may occur in from 6 to 12 hours or even longer.

According to Roholm (1937) post-mortem findings are congestion and haemorrhagic infiltrations of all organs, especially the lungs. The spleen is enlarged. The liver has a cloudy swelling and is of a yellow colour. The kidneys are swollen and oedematous and the stomach contains blood-stained fluid.

Wichmann and Dahle (1933) found 2 p.p.m. of fluorine in apples which had been treated with a fluorine insecticide.

Geiger (1936) records an outbreak of poisoning due to the ingestion of a mixture of sodium bicarbonate and sodium fluoride sold in bulk as sodium bicarbonate or baking soda which was responsible for poisoning in 20 reported instances, 3 of which terminated fatally.

Hanzlik (1936), referring to the above outbreak, remarks:

> The disionisation of calcium in the blood and tissues is undoubtedly responsible for the symptoms and tissue changes of the acute poisoning, and the more soluble the fluoride the more rapid the onset and more violent the symptoms.

Lidbeck, Hill, and Beeman (1943) mention a very serious outbreak of sodium fluoride poisoning affecting 263 persons with 47 deaths, which occurred in a hospital in Oregon, U.S.A. An insecticide powder containing sodium fluoride, for the extermination of cockroaches, was mistaken for powdered milk and used in making scrambled eggs, with disastrous results. Most of the fatal cases occurred in from 2 to 4 hours after ingestion of the food. One patient died 15 minutes after eating the poisonous scrambled eggs. The above outbreak shows that insecticides should not be stored in culinary offices.

Hauser (1945) records an example of sodium fluoride being mistaken for baking powder. This occurred in a large state institution in New Orleans.

One night 14 inmates became desperately ill, one of them dying. Food poisoning was immediately suspected because of the explosive character of the outbreak and many samples of food found in the kitchen were submitted for examination. Within a few hours sodium fluoride was found in one of the biscuits. The stomach of the dead woman contained the same poison. Samples of white powder in an unlabelled jar were found to be roach (cockroach) poison containing sodium fluoride. Subsequent investigation revealed that a new cook, during preparation of her first meal, mistook the roach poison for baking powder and used it in the biscuits.

Fluorine in Foods

McClure (1949) reviewed the distribution of fluorine in foods. Exclusive of drinking water the ordinary diet appears to provide 0·2 to 0·4 mg. of fluorine daily. The majority of foods found in the average diet contain from 0·2 to 0·3 p.p.m. or less fluorine. Tea and sea foods are exceptions, the former having upwards of 75 to 100 p.p.m. in the dry tea, whereas the amount present in sea foods is 5 to 15 p.p.m. Sea water, which is the source of the element in sea foods, averages 1·2 to 1·4 p.p.m.

Cow's milk contains about 0·1 to 0·2 of fluorine. If fluoride is added to the cow's ration or to the drinking water it does not effect the milk content. Fluorine in soil and water has little or no influence on the fluorine content of edible plant produce. Although the data are limited it appears that natural food-borne fluorine is largely available for body assimilation. The common cereals have been extensively analysed for fluorine with variable results, but very low (about 0·10 to 0·20 p.p.m.) in the fresh material.

Mackenzie (1950), discussing fluorine and water-borne diseases, recommends that a concentration of 1·5 p.p.m. fluorine should not be exceeded in public water supplies.

The Metallic Contamination Sub-Committee in their report of 1957 recommend that the Fluorine in Food Order, 1947, should be amended so as to set the following limits to the fluorine content of acidic phosphates intended for use in foods and of foods containing acidic phosphates:

	Articles of Food	*Maximum Fluorine Content*
(i)	Acidic phosphates	30 p.p.m.
(ii)	Any article of food (not included in items iii and iv below) containing acidic phosphates and intended for use in the composition or preparation of food.	30 p.p.m. of the acidic phosphates present.
(iii)	Baking powder, including golden raising powder.	15 p.p.m.
(iv)	Self-raising flour or any similar mixtures (not included in item iii above) containing a farinaceous substance and an acidic phosphate.	3 p.p.m.

Barium Carbonate

This substance is sometimes used for the extermination of rats and mice. It is a tasteless, odourless white powder, closely resembling articles used for culinary purposes. Morton (1945) recorded two outbreaks of food poisoning among British soldiers in an area of the Persia-Iraq Command, when barium carbonate was mistaken for flour.

The first in the sergeants' mess, involving 13 men, the second, a few days later, affecting 71 men in one unit. The vehicle in the first was marmalade tart, and in the second, treacle tart, and the outbreaks involved only those who had eaten tart. The onset in the first outbreak was about an hour, in the second, two hours after the meal, and the symptoms were more severe in the first outbreak. Chemical analysis of the pastry showed large quantities of barium carbonate; an average portion of marmalade tart contained about 15 gm., the treacle tart being less heavily contaminated.

Barium carbonate was found in the bulk stock of flour issued to these two messes. Its presence was due to the fact that a sack containing 4 lb. of barium carbonate, for use as a rat poison, had been placed in error in the flour store, and subsequently filled up with ordinary flour. It had been used only for pastry for the tarts. The poisonous agent is the barium chloride produced in the stomach by action of the gastric HCL. The symptoms were very consistent; an initial phase of acute gastro-enteritis with tingling sensation round the mouth and in the neck, followed by loss of tendon reflexes and various degrees of motor paralysis. In a few severe cases paralysis spread and became general. The action of the heart was disordered. Recovery was rapid and there were no deaths. The text book features of convulsions and clonic muscle contractions were absent. The author considers that barium carbonate for use as a rat poison should be coloured to prevent similar accidents. [Savage, 1946].

Budernik (1948) records the following cases of food poisoning by barium carbonate. The symptoms in the first (woman) patient were unusual as in addition to diarrhoea and vomiting there was stupor, spasms in arms and legs, and an irregular slow pulse rate of 28–32 per minute. The woman recovered but the pulse rate remained slow for some time. It was subsequently ascertained that some 4 weeks earlier 4 other families were attacked with similar symptoms, but one patient in addition suffered from paralysis of the lower jaw and another became unconscious; all recovered. The affected persons had eaten pudding made with meal. The meal was kept in a sack and found to contain barium carbonate in about 1·9 per cent. This would be absorbed as barium chloride. The barium carbonate was used as a poison for rats and mice and apparently was added to the meal inadvertently.

Food Contamination from Insecticides

Dubois (1950) discusses this matter as follows:

Within recent years a large number of new insecticides have come into extensive agricultural use. Nearly all are either chlorinated hydrocarbons and related chlorinated compounds or organic phosphorus-containing compounds. Since these materials are widely used on fruits, vegetables and forage for live stock, a consideration of the possible health hazards from contaminated foods has become of importance. The toxicologic information now available allows some estimation of the potential dangers, but further detailed studies on most of the new compounds are necessary in order to obtain an accurate evaluation of the possible health hazards.

In the case of the chlorinated hydrocarbons such as DDT, acute poisoning from single doses is rare. However, chronic poisoning from continued ingestion of the materials is demonstrable in animals, and the stability of these agents, together with their tendency towards accumulation and storage in the tissue lipoids, places emphasis on the necessity of avoiding food contamination through the use of excessive amounts of these materials. Information regarding the possible effects of ingestion of small quantities of these agents over long periods of time must await the results of further experimentation.

With organic, phosphorus-containing compounds now in use, acute poisoning has been the problem of major concern. The tendency of these insecticides to undergo rapid hydrolysis to non-toxic materials in the presence of moisture makes chronic poisoning unlikely. Thus far, no evidence indicating the possibility of poisoning through food contamination by the organic phosphates used as contact insecticides has been obtained.

The organic phosphates which are systemic insecticides by virtue of their ability to be absorbed by plants are more hazardous than the other organic phosphates, and it appears that these new agents should be employed with caution to prevent the possibility of accidental poisoning.

Kingsley Kay (1956) says in his article on 'Pesticides and Public Health':

The significance of the pesticide problem in public health is based upon the tremendous quantities of these potentially toxic materials now in use throughout the country. No other class of toxic chemical is so widely and freely used. Poisoning from agricultural chemicals has ranked second as causative agent in recent United States studies of systematic poisoning at the occupational level.

The rôle of trace amounts of synthetic organic pesticides which occur in present food supplies and the general atmosphere has not yet been delineated in relation to health. Accounts must be taken of recent animal studies in which biochemical and physiological effects have been found at levels of intake below those producing classical pathological effects. Health protection in use of pesticides must extend beyond labelling and the question of special instruction of the user, improved equipment and restriction of use is raised.

Administrative Measures and Advisory Pamphlets

Agriculture (Poisonous Substances) Act, 1952. This act affords protection to agricultural workers against the risk of poisoning by the following substances:

(*a*) dinitro-phenols and their salts.
(*b*) dinitro-substituted phenols and their salts.
(*c*) organo-phosphorus compounds.
(*d*) preparations or mixtures containing any of the substances mentioned above.

Sec. 9 (2) of the act allows of any other substance being added to the list if the Minister of Agriculture and Fisheries and the Secretary of State are satisfied that it would involve substantial risk of poisoning by agricultural workers.

In 1955, regulations under the above act (namely the Agriculture (Poisonous Substances) Regulations 1956) were made by the Minister of Agriculture and Fisheries and the Secretary for Scotland.

Leaflet APS/1 a summary of the provisions of the Agriculture (Poisonous Substances) Regulations 1956, revised May 1956, contains 'The Safe Use of Poisonous Chemical on the Farm'.

Advisory leaflet No. 374 has been issued by the Ministry of Agriculture and Fisheries on 'Precautions in the Use of Insecticides, Fungicides and Weed Killers'.

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Chapter XII

POISONOUS PLANTS

IT has long been known that certain plants, when consumed, are poisonous to human beings. Poisoning of this kind is usually accidental, the particular plant being gathered and eaten in ignorance of its nature, or in a mistake for some harmless variety. The majority of cases occur among children who are especially liable to eat the attractive leaves or berries, including seeds of the peach and bitter almonds. The number of such plants in this country is comparatively few. Some are rare, others are only noxious at certain periods of the year. In a few instances parts of the plants only are poisonous.

The most common are the following: Hemlock (*Conium maculatum* L.); Cowbane or Water Hemlock (*Circuta virosa* L.); Water Dropwort (*Oenanthe crocata* L.); Monkshood or Aconite (*Aconitum napellus* L.); Deadly Nightshade (*Atropa belladonna* L.); Foxglove (*Digitalis purpurea* L.); Henbane (*Hyoscyamus niger* L.); Black Hellebore (*Helleborus niger* L.); Bittersweet or Woody Nightshade (*Solanum dulcamara* L.); Fool's Parsley (*Aethusa cynapium* L.); Bryony (*Bryonia dioica* L.); Laburnum (*Cytisus laburnum* L.); Black Nightshade (*Solanum nigrum* L.); Spurge Laurel (*Daphne laureola* L.); Annual Mercury (*Mercurialis annua* L.); Dog's Mercury (*Mercurialis perennis* L.); Mezereon (*Daphne mezereum* L.); Black Bryony (*Tamus communis* L.).

The above plants contain poisonous substances or alkaloids, the chief being strychnine, atropine, coniine, aconitine, hyoscyamine, scopolamine, solanine, and cytisine.

HEMLOCK (*Conium maculatum* L.), SPOTTED HEMLOCK

This noxious biennial plant, which grows on waste land, banks, hedgerows, and by roadsides and streams, is widely distributed and flourishes especially in Yorkshire and the North of England. The tall (3 feet to 5 feet high), glossy, smooth hollow stem is branched and marked with purplish-red spots. The large leaves somewhat resemble parsley and the small white flowers, which appear during June and July, are arranged in umbels (as the ribs of an umbrella). When not flowering hemlock may be recognized by the appearance of the fruit. Each carpel is nearly globular with

5 permanent broad flat ribs, with single vittas under the furrows. The plant has a strong disagreeable mousy odour when bruised or crushed.

Hemlock owes its poisonous properties to a number of alkaloids, the chief of which are coniine, methylconiine, coniceine, and conhydrine. Coniine is present in the leaves of the plant, but later is found in the fruit and seeds. Farr and Wright (1887–8) give percentages of alkaloids in the various parts of the plant: stem, 0·0–0·6; leaves, 0·03–0·18; flowers, 0·9–0·24; green fruit, 0·73–0·98; range of fruits collected in England, 1·05–3·06. Coniine was isolated by Giesecke (1827) and was the first alkaloid to be synthesized. Coniine is a colourless alkaline liquid with a penetrating odour and burning taste. All the hemlock alkaloids are poisonous, causing paralysis of the motor nerve terminations. Stimulation is followed by depression of the central nervous system. Nausea and vomiting occur at an early stage. Respiration is accelerated at first but gradually becomes slower, laboured, and finally ceases. The heart continues to beat even when consciousness has disappeared.

The pale yellow tapering root when bruised has an odour like the parsnip. It is less poisonous, however, than other parts of the plant, but this varies with the time of the year. It is, however, especially noxious in the first season's growth.

Many cases of poisoning have been recorded through mistaking hemlock leaves for parsley when eaten in salad or soup; also through the consumption of the root and seeds, the latter being mistaken for, and accidentally mixed with aniseeds (Galtier, 1885). The symptoms of hemlock poisoning are, burning sensation in nose and throat, inability to swallow, languor, drowsiness, twitching, tremors, and staggering gait, with muscular weakness, stiffness and rigidity of the legs due to partial paralysis of the peripheral nerves. Occasionally vomiting with profuse salivation occur and the pupils of the eyes become dilated. Stupor and coma may supervene; death results from respiratory failure.

The noxious property of hemlock was well known to the ancients, and history relates that the juice from the plant was administered to the Greek philosopher, Socrates. The Anglo-Saxons were aware that the plant had certain properties and used it in their medicines.

According to Henslow (1901),

That the poisonous property is not destroyed by boiling is confirmed by a case of two soldiers who collected herbs for boiling with bacon.

They partook of the broth, and then of the herbs and bacon. They died in about three hours.

In another instance children were poisoned by blowing whistles made from twigs of spotted hemlock.

Wulsten (1926) records the poisoning of 2 children who had picked and eaten hemlock, which they found growing by the roadside at Burg, near Magdeburg. One child died in convulsions 3 hours after eating the plant, but the other child recovered and was discharged from hospital 4 days later. The symptoms in these cases were, convulsions, cramp of the entire musculature and vomiting, dilated pupils and no reaction, skin cold, moist, and markedly cyanotic.

Cowbane or Water Hemlock (*Circuta virosa* L.)

Cowbane, which is a fairly common plant in England, Scotland, and Ireland, flourishes in damp situations, such as marshes, ditches, edges of ponds, lakes, and rivers, and grows to a height of 3 to 4 feet. The stem is sometimes reddish in colour, stout, hollow, furrowed, and branched not unlike celery with large bright green leaves about 1 to $1\frac{1}{4}$ inches long, the segments of which are long and narrow. The plant has a disagreeable mouse-like odour and all parts (fresh or in the dried state) are poisonous to man, especially the root-stock.

The seeds resemble anise and the small white flowers appear from July to August. The short tapering root, commonly known in some districts by the name of 'five-finger root' is fleshy, hollow, and contains a yellow juice. The root is often mistaken for wild parsnip or Jerusalem artichoke. The consumption of a small portion of the root or some of the leaves causes a burning pain in the stomach, vomiting, giddiness, convulsions, and sometimes death. Its poisonous properties are due to toxins (Svagr, 1923) and the name often given to the poison is 'circutoxin'.

Many instances incriminating cowbane are on record. In 1901 a party of boys were camping out on an island in the Firth of Clyde and 24 of them were poisoned by eating this plant.

Gompertz (1926) reported an outbreak in a Connecticut institution of 17 simultaneous cases in boys who had eaten roots, leaves, or flowers growing near their playground. Less than 2 hours later all were violently ill, with vomiting and convulsions. They received medical aid at once and entirely recovered the next day, with no lasting effects from the illness.

WATER DROPWORT (*Oenanthe crocata* L.)

This is another member of the hemlock family and one of the most common poisonous plants in this country. It grows in ditches, and marshes, on the banks of rivers or streams, and in other damp places, sometimes actually in the water. All parts of this perennial plant are poisonous, especially the fleshy, juicy, yellow spindle-shaped root, which is the chief seat of the poison.

The tall stem (3 to 5 feet in height) is grooved, branched, and hollow with large compound leaves having divided leaflets which resemble wild celery, especially when the plant is not in flower. The leaves when crushed exude a pungent smell like that of celery. The cluster of small yellowish-white flowers appear during July. The fruit is narrow and oblong. The juice of the stem and root becomes yellow on exposure to the air. The root is sometimes mistaken for the parsnip. The toxic material contained in the plant and root is non-alkaloidal and its constitution has not been definitely established but it is sometimes referred to as 'oenanthotoxin'.

Sowerby and Johnson (1861) record the poisoning near Woolwich of 17 convicts, who gathered and ate the weed, mistaking it for celery and parsnips. Nine suffered from convulsions and 6 died. The symptoms were tetanus, delirium, and insanity.

Holmes (1902) gave it as his opinion that water dropwort is the most dangerous and virulently poisonous of all our native species. Its effects are rapid and fatal within a few hours after the ingestion of a small piece of the root.

In November 1945, two Italian prisoners of war died through eating the root of this plant. One of the men was seen with a piece in his hand. At the autopsy the stomachs of both were found to contain the macerated root which was identified as water dropwort.

At Barrow-in-Furness in September 1947, 5 children were given food by their mother and sent into the country for a half day's holiday. Some hours later, a man passing a field heard groans and other distressing sounds. Upon investigation he found 4 of the children in convulsions and the other child screaming with fright. One was seen to be holding part of the plant in his hand and other pieces were on the ground nearby. Two of the children died in hospital the same night and one the following morning. The other two children recovered. At the post-mortem examination pieces of the root of the plant were found in the stomachs of the children. The pathologist confirmed that the ingestion of the root of water dropwort was the cause of the deaths.

Monkshood (*Aconitum napellus* L.), Aconite, Wolf's Bane, or Blue Rocket

This perennial plant, the poisonous nature of which was well known to the ancients, grows on moist pastures, in thickets, is common in the West of England and in the river valleys of Yorkshire and South Wales. The cultivated plant is used for medicinal purposes.

The plant is about 3 or 4 feet high and grows in circular patches. In the spring it is recognized by the glossy bright green, deeply-fingered leaves, which appear before the tall leafy stems. The large dark blue or purple helmet-shaped (monk's cowl) flowers, variegated with white, are on erect short stalks forming a dense terminal raceme. The upper helmet sepal at first conceals the lateral ones, but it is later thrown back. The flowers appear from July to September. The numerous seeds are three-sided, irregularly twisted, wrinkled, dark brown in colour and about one sixth of an inch in length. The root is conical or spindle-shaped, pale brown in colour on the outside and white inside and of a fleshy nature, which distinguishes it from the cylindrical pungent root of the horse-radish, with which it is often confounded, resulting in cases of poisoning. The leaves also have been eaten as a salad with fatal results.

All parts of the plant are noxious. Symptoms of poisoning from aconite appear quickly and the course of the illness is rapid. There is tingling of lips and tongue, numbness in mouth and face, burning sensation in throat and stomach, salivation and nausea which is usually followed by retching, vomiting, and grinding of the teeth. Numbness and tingling occur in fingers and legs and there is dilation of the pupils, difficulty in swallowing and sometimes talking. The pulse is slow, feeble, and irregular and respiration shallow and rapid. The surface of the body is cold and moist, and the face bloodless. Death may occur in three or four hours from syncope or respiratory failure.

Sowerby and Johnson (1861) say:

> Its frequency in the garden and the careless manner in which its deadly roots are often distributed have induced us to place it at the head of our list of British poisonous plants. The recent accident in Scotland, where 3 persons died in consequence of the roots of the monkshood being brought in by a boy from the garden as horse-radish and used by the cook, unconsciously, in preparing sauce for beef, added to many others of a similar kind, ought to render gardeners cautious in planting and teach them to avoid placing this and other poisonous herbs in the vicinity of those employed for culinary purposes.

FIG. 5. MONKSHOOD.

Henslow (1901) remarks: 'So acrid is the poison that the juice applied to a wounded finger affected the whole system; not only causing pains in the limbs, but a sense of suffocation and syncope.' The virulent properties of aconite (active principle, aconitine), however, depend to some extent on the age of the plant and the climate in which it is grown.

Deadly Nightshade (*Atropa belladonna* L.)

Sometimes called Dwale, Barnewort, or Naughty Man's Cherry, this is a well-known and widely distributed perennial plant growing in chalky soils, on waste ground, borders of fields and in hedgebanks, especially on the North and South Downs and the Cotswolds. It is extremely poisonous (acrid narcotic) to man, but like many other noxious plants there are seasonal variations. The cultivated is less poisonous than the wild variety.

Its noxious properties and fatal effects seem to have been long known, and it is supposed that Dwale was the poisonous plant which occasioned such disastrous consequences to Roman troops under Mark Antony at their retreat from the Parthians.

The downy leaves of deadly nightshade are large, oval, pointed, and arranged in pairs, consisting of a large and a small leaf. The bell-shaped flowers, each about 1 inch in length with 5 broad short lobes, are of a dull purplish-blue colour. They are on short peduncles situated in the forks of the stem or in the axils of the leaves. The flowers appear from June to August. Just before flowering the stout whitish-coloured fleshy root, which is the most noxious part of the plant (although all parts are poisonous) is richer in the poisonous principles, atropine, hyoscyamine, and scopolamine, than after flowering. In August and September the shiny, ripe, juicy purplish-black berries, containing very small rough kidney-shaped seeds, are tempting in appearance and have a somewhat sweetish taste. They resemble small cherries with deep central furrows and are especially attractive to children, who are more susceptible than adults to the effects of the poison. The consumption of three or four of the berries causes great excitement, rapid pulse and respiration, dryness of the mouth and throat, double vision, nausea, vomiting, delirium and hallucinations, the speech is thick and incoherent. In fatal cases, periods of drowsiness or stupor interrupt the delirium, but eventually the coma becomes profound, the face livid, and the skin cold. Death (which may take place within 24 hours) usually occurs from heart failure.

Henslow (1901) records a remarkable outbreak of poisoning

which occurred in 1846, due to berries of deadly night-shade being sold by ignorant dealers in the streets of London as edible fruit; two persons died.

A curious case of poisoning by 'belladonna leaves' is described by Hope (1921)—quoted by Savage and Bruce White (1925):

On 8th May, after partaking of roast stuffed breast of mutton and potatoes, a lady and her two daughters became ill. Their symptoms were dryness of the mouth, giddiness, weakness of limbs, and disturbance of vision and started about ten minutes after eating the food. Belladonna poisoning was diagnosed. The mutton was stuffed with breadcrumbs, salt, pepper, mint, sage and onions. The chemical examination showed that the portions of meat and sage stuffing examined weighed 3 oz. and contained $\frac{1}{40}$ grain of atropine. The dried herbs were obtained from the district of Evesham, and inquiry of the Worcestershire County Medical Officer elicited that the belladonna plant was at one time largely grown in the vicinity of Evesham, but the industry had entirely ceased since 1918. Although its cultivation had ceased and the roots had as far as possible been destroyed, odd plants still continued to come up as the root is very difficult to eradicate. Evidently some belladonna leaves had become mixed with the herbs sent.

During 1948, a number of cases of poisoning occurred among children in the south of England who had eaten the berries of the deadly nightshade. Five cases are recorded by Minors (1948) who states:

The important features in these five cases of poisoning appear to be: (1) The prolonged period between the ingestion of the berries and the appearance of the symptoms. (2) The absence of any fever or respiratory depression and the prominence of the hallucination. (3) The significance of 'raisins' in the vomit—so unlike fresh deadly nightshade berries—might not have been appreciated in a case where no history of eating berries was obtainable. (4) The necessity for administering an emetic: many of the berries would have blocked the largest size of stomach tube.

It has been suggested that some special instruction on the subject of poisonous berries might be given to school children.

FOXGLOVE (*Digitalis purpurea* L.), Throatwort or Deadman's Bells

This handsome flowering biennial plant grows in cleared woodlands, copses, and hedgerows on siliceous soils and is common in most parts of England. From ancient times it has been recognized as being one of our most poisonous herbs. The erect stem is 3 to 4 feet in height and covered with grey down. The large, downy, dull green leaves, which have a faint odour and a bitter nauseous taste, terminate in a long one-sided bunch of bell-shaped purple flowers which open between July and September. All parts

of the plant are poisonous, especially the small brown seeds, either fresh or dried. The leaves are more noxious before the plant flowers. Foxglove is also cultivated but this is less poisonous than the wild variety. Large doses of the poison cause vomiting, purging, fainting, and may prove fatal.

Foxglove is the source of the well-known drug digitalis and contains four distinct active principles: digitalein, digitonin, digitoxin, and digitalin, which are not destroyed by drying or boiling the plant.

Henbane (*Hyoscyamus niger* L.) or Hen-bell

An annual or biennial, erect, coarse, hairy plant which attains a height of about 1 to 2 feet. It grows in waste sandy soils, in stony situations, under hedges, and on roadsides. The plant is fairly common in England, Wales, Southern Scotland, and Ireland. The large dark, greyish-green oblong toothed leaves are thick, hairy, and woolly. The odour of the fresh leaves produces giddiness and stupor. The five-lobed funnel-shaped flowers, which appear in June, July and August, are of a dirty yellow colour (paler towards the edges) with purple veins, and are arranged in rows all along one side of the stem. The flowers are in time replaced by greyish seed capsules.

Henbane seeds have been eaten by children with serious results. Twenty seeds cause grave results in man.

The whole plant, which is sticky to the touch, is poisonous and has a strong offensive odour. It contains the alkaloids scopolamine and hyoscyamine which are powerful narcotics and present in greater quantity at the time when the seeds are ripening. The large whitish thick branching root is sometimes mistaken for parsnips.

Dr. Houlton records that the whole of the inmates of a monastery were poisoned by using the root instead of chicory. It produced hallucinations, but no deaths.

Among the symptoms of poisoning following the consumption of henbane and its seeds are: flushed face, dilated pupils and bright shining eyes, dryness of throat, marked disturbance of vision, and hallucinations. The patient is talkative and may become violent. Later, there is drowsiness followed by coma.

Bitter Sweet or Woody Nightshade (*Solanum dulcamara* L.), Felon Wood, Felon Wort or Mortal

This climbing and trailing perennial July plant, which is found throughout England and Ireland and sometimes in Scotland,

FIG. 6. BITTER SWEET.

usually grows in moist and shady positions in woods and hedges. It climbs over and around hedges by means of claspers on the branches, sometimes to a height of 5 to 6 feet. The dark-green, pointed leaves, about 2 to 3 inches long, have clusters of small purplish-blue flowers with yellow anthers in loose lateral peduncles shorter than the leaves; these appear from June to September. The glistening red berries, which are attractive to children, are egg-shaped and hang on the branches during autumn and early winter. The stem of the plant is at first bitter and then sweet to the taste. The stem, leaves, and berries are poisonous to man. The toxic principle is solanine.

Alexander, Forbes, and Hawkins (1948) record the case of a child (aged 9) who died as a result of eating the red berries of the woody nightshade (solanine poisoning). 'According to Reil (1857) Solanine destroys life by producing paralysis of the muscles of the chest. It is a slow-acting poison, and as far as we know has not yet been isolated from the vomit or stomach washings of suspected cases. It differs from atropine (deadly nightshade) and hyoscyamus (henbane) in not producing stupor or delirium, dilatation of the pupils, sphincter paralysis or pyrexia.' The early symptoms are abdominal pain, vomiting, diarrhoea, and depression.

Fool's Parsley (*Aethusa cynapium* L.), Fool's Cicely, Dog's Parsley, False Parsley

An annual plant which belongs to the hemlock family and grows in gardens, fields, and hedges, all over the British Isles. It reaches a height of 6 inches to 2 feet. The stem is hollow, branches marked with fine lines, and the very dark glossy wedge-shaped green leaves somewhat resemble common parsley. Small clusters of white flowers appear in July to September. It may be distinguished from similar plants by three slender leaflets hanging from each of the small clusters of flowers forming the general cluster. When bruised the plant gives off an unpleasant odour. All parts of the plant are poisonous to man. Accidents have been caused by the consumption of the leaves and root in mistake for parsley, radishes, or turnips. A case occurred in Germany a few years ago where the leaves were put into soup in mistake for parsley. Vomiting and diarrhoea followed, the lower jaw became fixed (tetanus) and death occurred within 24 hours.

Saltmann (1931) recorded the poisoning of a family after partaking of potato soup which had been accidently flavoured with Fool's Parsley. The symptoms were nausea, abdominal pains,

Plate 17. Henbane

(*a*) Hemlock

(*b*) Fool's Parsley

(*c*) Cowbane or Water Hemlock

(*d*) Cowbane Tubers

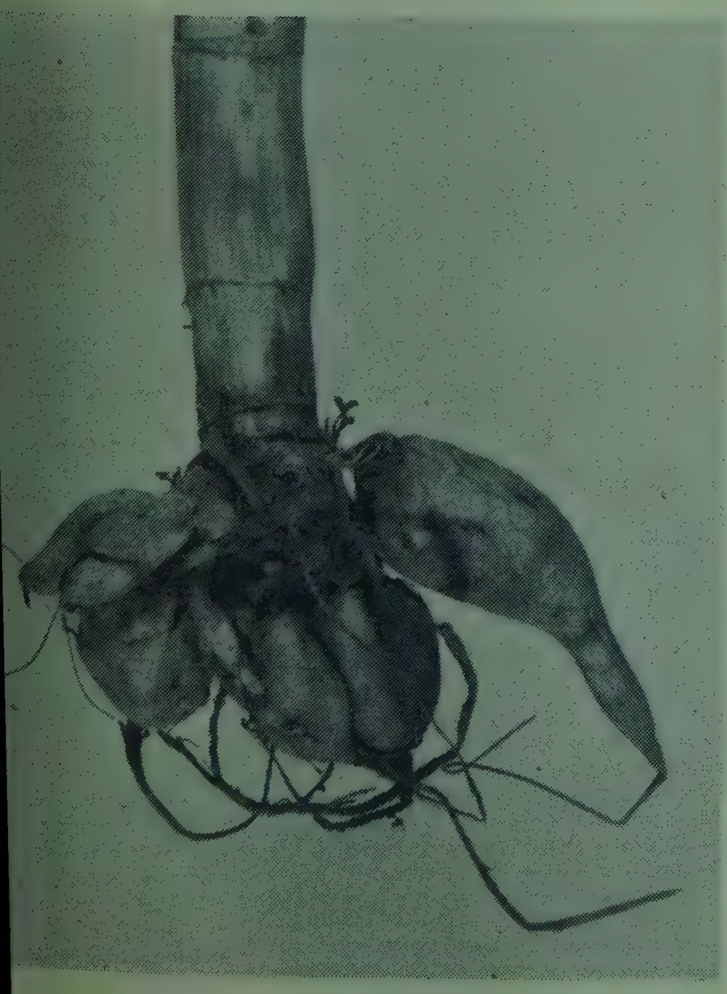
PLATE 18

Plate 19

(*a*) Deadly Nightshade (above)

(*b*) Water Dropwort Root (below)

(*c*) Foxglove (right)

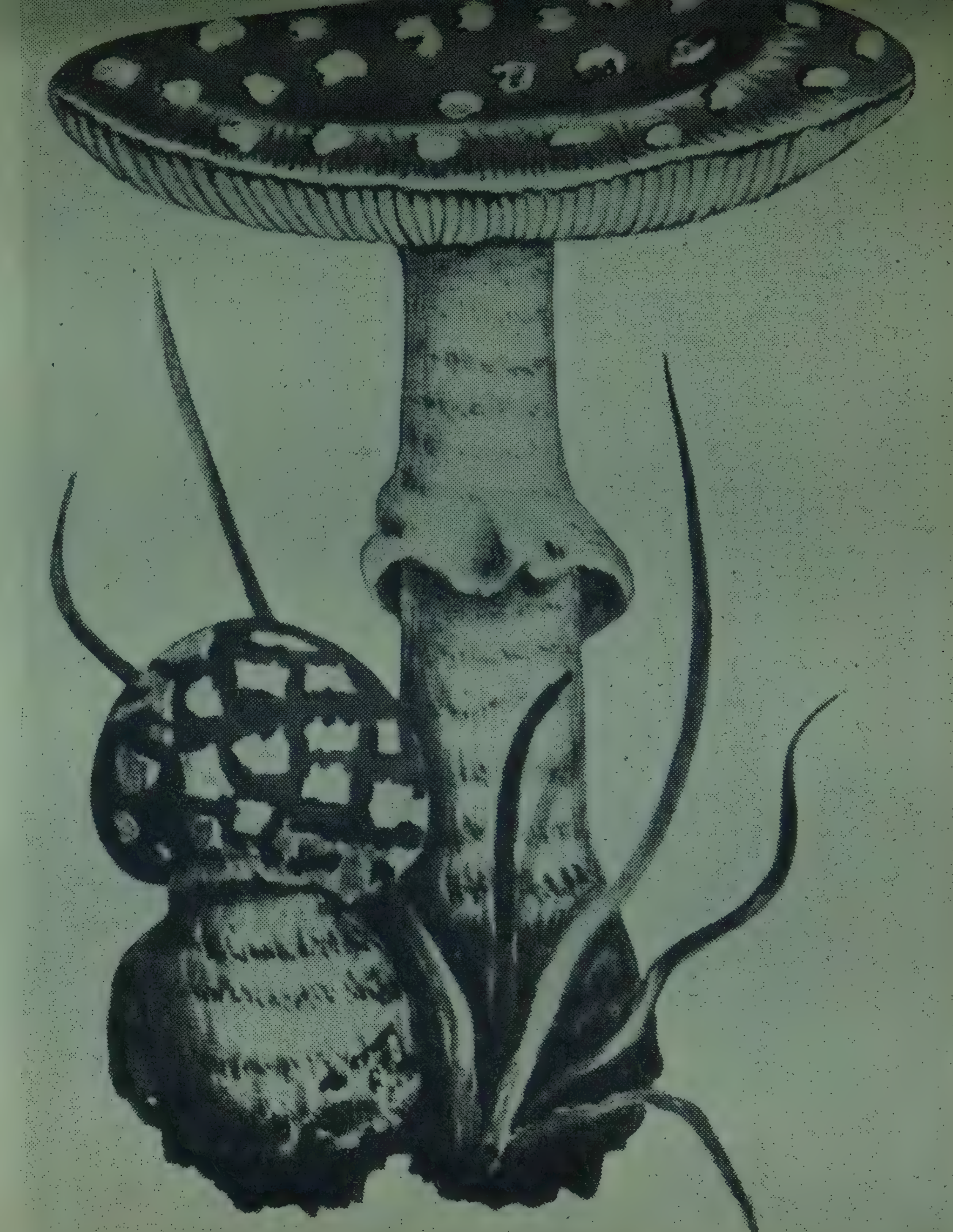


(*a*) Common Mushroom

(*b*) Verdigris Agaric

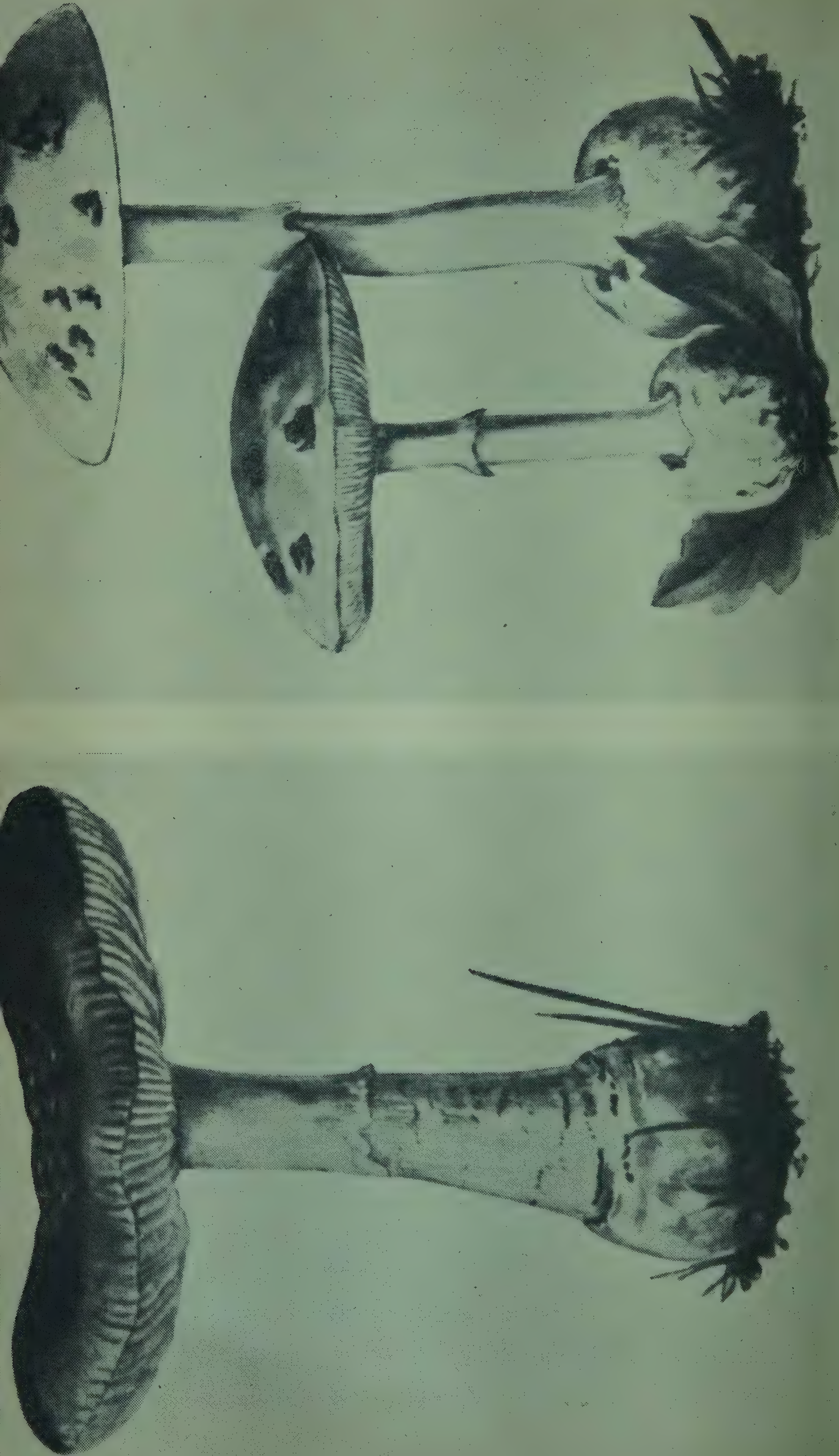
PLATE 20

(*a*) Death Cap



(*b*) Fly Agaric

PLATE 21




(*a*) Purple Agaric


(*b*) Yellow Staining Mushroom

PLATE 23

The poisonous principle is the alkaloid cytisine. The symptoms of poisoning, which appear about half an hour after eating the pods or seeds are nausea, vomiting, drowsiness, mydriasis, excessive rapidity of the heart's action, muscular weakness, and incoordination. In severe cases there are convulsions followed by respiratory paralysis and death.

FIG. 7. BRYONY.

FIG. 8. BLACK NIGHTSHADE.

Mitchell (Edinburgh, 1951) records 10 cases of children (ages 3 to 10 years) poisoned through eating the pods or seeds of laburnum during the months of July, August, or September. All the children recovered within 24 hours. Mitchell remarks:

> The poisonous effects of laburnum have been known for many years, especially in the North of Scotland where its narcotic and emetic properties were common knowledge among the rural population a hundred years ago.

He also quotes the following references: Christen (1949); Wheelhouse (1870); Joll (1879); De la Motte (1880); Somers (1883); Tomlinson and McNaughton (1895).

BLACK NIGHTSHADE (*Solanum nigrum* L.)

An annual (or biennial) plant. It is common in England and is also found in Scotland and Ireland. It grows on or near walls, on

waste land, at the sides of hedges, fields, on sea beaches, and in cultivated gardens, where it sometimes becames a troublesome weed. The branched stem is upright, round, hollow, and the plant attains a height of from 1 to 2 feet. The juicy leaves are soft, smooth, oval, pointed, and unevenly indented at the edges. The root is whitish and sometimes woody. Near the top of the stalk are clusters of small white flowers which expand in summer and early autumn. These give way to pendulous berries, which ripen in October, and somewhat resemble black currants. At first their colour is green, turning later from reddish-black to black. They contain a greenish juice and white seeds. The plant has a disagreeable odour and contains the alkaloid solanine, chiefly in the berries, and in the leaves and stem. The amount is said to vary with conditions of climate, season of the year, and kind of soil. Henslow records the fact that children have been poisoned after eating the berries, which cause nausea, vomiting, colic, purging, and convulsions.

Spurge Laurel (*Daphne laureola* L.), Copse Laurel, Wood Laurel, or Dwarf Bay

This erect, glabrous, evergreen shrub, which grows to a height of 2 to 4 feet, is found in banks, hedgerows, woods, and copses, especially where the soil is stiff or clayey, and abounds in Yorkshire, Durham, and some of the southern counties of England. It is divided near the top into several branches. Among the oblong smooth shining dark leaves, and near the top of the branches, are clusters of small yellowish-green flowers (February to May) having an unpleasant smell, which in time are replaced by bluish-black berries. The leaves, berries, and bark have a bitter taste and are poisonous, narcotic, and purgative. The toxic principle is mezerinic acid. Henslow remarks that eating the acrid bark has proved fatal to children. The symptoms of poisoning are a burning of lips, mouth, and throat.

Dog's Mercury (*Mercurialis perennis* L.), Herb Mercury, Wild Spinach or Kentish Balsam

This poisonous perennial hairy plant, a member of the spurge family and abundant in England and Scotland, grows to a height of about 1 foot in shady situations under hedges and bushes and in woods. It has a disagreeable odour. The long, broad-pointed dark rough green leaves are near the top of the plant and the stalk is round, thick and whitish in colour. The small fertile green flowers

FIG. 9. SPURGE LAUREL.

slender axillary peduncles appear in March, April, and May, on almost before the leaves are fully developed. The male flowers are in small clusters, the females single or in pairs. The fruit is a two-lobed capsule which is covered with hairs and contains two warted seeds. The root is slender and creeping. The plant contains a milky, poisonous sap. The active principle is Mercurialine—oil of euphorbia, which causes nervous symptoms if swallowed and acts as an emetic and strong purgative. Cases have been recorded where the plant (even when boiled) has had fatal results.

MEZEREON (*Daphne mezereum* L.), Dwarf Bay Tree, Spurge Olive

This small glabrous shrub with erect branches, which grows to a height of about 1 to 3 feet, is found in copses, woods, gardens, and shrubberies in the southern counties of England. Clusters of pink or purple sweet-scented flowers appear around the stem in early spring, terminated by a tuft of delicate green leaves. The poisonous scarlet berries (about the size of red currants) if eaten produce thirst and a sense of heat in the mouth and stomach, accompanied by prostration and fever. Fatal cases due to the consumption of the berries by children have been recorded. The toxic principle is said to be mezerinic acid, which is not destroyed by drying or storage.

BLACK BRYONY (*Tamus communis* L.)

This plant grows in hedgerows, woodlands, and copses in many parts of England and is sometimes found in Scotland. It is a climber, having long trailing stems many feet in length, which hang in festoons over the surrounding hedges. The bright, glossy, heart-shaped, pointed leaves are on long stalks and the small greenish-yellow flowers, which spring from the axils of the leaves, appear in the spring and early summer. Numerous green or scarlet berries (resembling those of ordinary white bryony) are conspicuous in the late summer and autumn. The berries, if consumed, cause vomiting, intestinal pains, paralysis, sometimes with fatal results. The large fleshy dark-coloured root has an acrid taste, possesses purgative properties, and is poisonous to man.

RHUBARB

Rhubarb has frequently been the cause of poisoning, both at home and abroad. The stalks and leaves contain 0·2 to 0·4 per cent of oxalic acid.

FIG. 10. DOG'S MERCURY.

FIG. 11. ANNUAL MERCURY.

FIG. 12. MEZEREON.

FIG. 13. BLACK BRYONY.

Rosenau and his associates at the Harvard Medical School recorded a small outbreak due to rhubarb leaves to illustrate fallacies in diagnosis of 'ptomaine poisoning'.

A female, aged 58, ate about a half-peck of cooked rhubarb leaves (tops) on 14th May, 1917, at 4 p.m. Also took much of the water in which the leaves were cooked. She was sick all night; started to vomit about 3 a.m. on the morning of the 15th. At the same time diarrhoea commenced and continued during the night, but bowels did not move again until the time of death. Patient vomited throughout the entire illness, was very thirsty, and drank a great deal of water. Temperature normal. Patient had had chronic pains in abdomen for many years, which were much intensified during this period. Died in ten days. Rigor mortis did not set in until 30 hours after death. Bacteriological examination of material obtained post-mortem by rectal swab showed that the case was complicated with dysentry.

A brother-in-law of the patient ate some of the greens and was sick all night, but recovered. A sister ate a very small amount of the greens, and had but slight malaise. Diagnosis: Oxalic acid poisoning.

Burton (1910), who observed two cases of poisoning caused by eating stewed rhubarb, stated that some people may be more susceptible and others more resistant to oxalic acid poisoning. The patient suffered from diarrhoea, prostration, and purging. During the food scarcity in the First World War the use of the leaves was recommended as a substitute for green vegetables (*Lancet*, 1917). The recommendation was, however, soon withdrawn as a number of deaths resulted from poisoning. In 1917 a warning was issued against using soda when cooking rhubarb. Most of the cases of poisoning have been the result of the use of the leaf stalks.

Benson (1919) records an outbreak of canned rhubarb poisoning. Nine cases occurred in one family. All were violently ill and two had convulsions. The symptoms appeared about 2 hours after the consumption of the rhubarb, all the patients recovered.

Tanner (1933) remarks:

In certain parts of France rhubarb leaves are eaten in place of spinach. This custom has caused some serious cases of poisoning. The symptoms appeared in a few hours after the meal, and included pains in the stomach, diarrhoea and cloudy urine of a mahogany colour with large amounts of albumin and cells.

The chief symptoms of oxalic acid (oxalates) poisoning are local irritation and constriction of mouth and throat. There is abdominal pain, sometimes severe with cramps, diarrhoea, and vomiting, and weak pulse. Muscular tremors may be marked, with

generalized convulsions and depression followed by coma. Death may result from shock and convulsions.

Poisoning by the ingestion of bread made from wheat contaminated by the seeds of certain weeds occurred in South Africa; it was known as 'bread' poisoning. The suggestion was that the seeds found their way into the wheat when the threshing machines and mills were not fitted with efficient winnowing and sieving apparatus. In most cases the weed was identified as senecio, commonly known as ragwort, of which there are several species. Ragwort is a scheduled noxious weed in this country, and its destruction may be made compulsory under an order of the County Agricultural Committee.

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Chapter XIII

EDIBLE AND POISONOUS FUNGI

FROM the earliest times we find records that man regarded fungi as a possible source of food. History relates that the eating of them was a popular custom among the ancient civilized peoples. The Greeks and Romans at their feasts and banquets indulged in the consumption of many different varieties, the boleti being in special favour, truffles coming next in esteem.

One of the earliest attempts to classify fungi was made by the Greek physician Dioscorides Pedanius (*De materia medica*), who divided them into the edible and the poisonous. He considered, however, that certain edible species were very indigestible and suggested they should be consumed with other substances and liquids; and as a precautionary measure advocated the use of an emetic after the meal. There are numerous references in classical writings to the ways of distinguishing these two groups, and several writers recommended certain simple tests to distinguish edible from poisonous fungi. These have been found, however, to be quite unreliable and dangerous and their use for this purpose has often resulted in serious illness which sometimes terminated fatally.

Ford (1909) who compiled an interesting historical review of the subject, carried out in conjunction with his co-workers at Johns Hopkins in Baltimore valuable work on this type of food poisoning.

Edible fungi now constitute an important article of diet both in this country and abroad. The field mushroom (*Psalliota campestris*), horse mushroom (*Psalliota arvensis*), and especially the cultivated variety, are at times in considerable demand, both for edible and canning purposes. Of late years the cultivation of mushrooms commercially has become a recognized industry. Their food value, however, from a scientific point of view is relatively small. They contain about 90 per cent moisture. The chemical value of some species of fresh edible fungi may be classed with certain vegetables in their use as additions to the ordinary diet.

Merrill (1916, quoted by Jordan, 1931) recorded an instance where a poisonous species grew in a mushroom bed almost to the exclusion of the common cultivated variety and was eaten by 5

members of the grower's family with almost fatal results. Poisonous mushrooms may apparently develop from commercial spawn, and growers must be careful to eat or sell from the beds only the common mushroom with white cap and pink gills.

Regarding fungi poisoning in the United States Jordan (1931) remarks:

> There is reason to believe that mushroom (or 'toadstool') intoxication in the United States has occurred with greater frequency of late years, partly on account of the generally increasing use of mushrooms as food and the consequently greater liability to mistake, and partly on account of the increase in immigration from mushroom-eating communities of Southern Europe.

The cultivation of the mushroom began in France and was described by Tournefort in 1707; later, very large quantities of edible fungi of several varieties were grown near Paris in underground caves some miles in length.

Paulet, who made a study of the incidence of fungi poisoning, records that in the environs of Paris between 1749 and 1788 there were at least 100 deaths. Guillaud (1885) believed that about 100 deaths were thus caused annually in the south of France. Ford (1923) mentions that approximately 1,000 cases with 318 deaths and 171 cases with 49 deaths had been recorded in the medical literature of France, and Germany Austria respectively. The same writer reported the occurrence of at least 217 authentic cases with 91 deaths in the United States during the previous 30 years. Out of more than a thousand species of mushrooms described in the United States over 80 were proved definitely poisonous (Jordan, 1931).

The study of edible and poisonous fungi has been pursued by many workers, and French scientists have done much to increase our knowledge of the subject. It is recorded that the first systematic investigation was carried out in 1791 by Bulliard, a French mycologist; he gave the name of 'Destroying Angel' to the species *Amanita verna.*

In this country many books and monographs have been written from time to time on the subject. In 1832, James Sowerby, Junr., compiled an illustrated work on mushrooms and champignons. In 1891 and in 1894 Cook published his books on *Edible and Poisonous Mushrooms* in which he mentions 22 species of the poisonous variety.

In 1910 the Board of Agriculture issued a small illustrated handbook *Edible and Poisonous Fungi.* This excellent work, of

which several editions have since been published by the Ministry of Agriculture, Fisheries and Food (*Bulletin*, No. 23) contains coloured illustrations and detailed descriptions of 27 varieties, 18 edible and 9 poisonous. Contrary to general belief, the number of fungi in this country which have poisonous properties are comparatively few.

During the late summer and early autumn (mushroom season) cases of poisoning frequently occur as a result of persons confusing the edible with the poisonous. The degree and severity of the resulting illness is in proportion to the quantity of ingested poison, as the poisonous properties are chemical in nature and vary in potency in the different species. Idiosyncrasy of the individual plays a part in fungi poisoning. Even the ordinary field mushroom disagrees with some persons and may cause intestinal disturbance, especially if not fresh or badly cooked. Price (1927) attributes four cases of illness, following a meal at which mushrooms were eaten, to the fact that they were decomposed and that some of them had been frozen.

During the First World War, when there was a shortage of foodstuffs in Germany and Austria, the incidence of this type of poisoning greatly increased. Roch (1916) recorded numerous outbreaks in the districts around Geneva. In 1921, owing to the increasing number of cases and deaths in France, a publicity campaign was started. The Pasteur Institute exhibited different species of edible and poisonous fungi and made known the precautionary and other measures to be adopted to combat this type of food poisoning. In some foreign countries laws exist regulating the sale of all fungi, and those retailed for consumption are subjected to an official inspection. In America, the whole subject has been carefully investigated by several workers, including Ford, Abel, Bronsen, Patterson, and Charles, McIlvaine, and Schlesinger, and as a result, much useful information has been forthcoming. Ford (1923) divided the poisonous fungi into groups:

(A) Gastro-intestinalis: those causing gastro-intestinal disturbances of a more or less violent character, but rarely with fatal results. The species chiefly concerned are: Boletus satanas, Lactarius torminosus, Russula emetica, Entoloma lividum, Lepiota morgani and B. miniato-olivaceus.

(B) Choleriformis: those producing the degenerative changes in the internal organs and tissues, loss of weight together with initial gastro-intestinal symptoms followed by violent pain, delirium and coma, with high mortality. Species concerned: Amanita phalloides, A. verna, Pholiota autumnalis and Hygrophorus conicus.

(C) Nervosus: those in which the poisons act on the nerve centres causing profuse perspiration or salivation, followed by delirium, hallucinations, convulsions and coma. In the early stages there is violent gastro-intestinal disturbance. There are, however, many mild cases of this type. Among the many species concerned are Amanita muscaria, A. pantherina, Clitocybe illudens, C. sudorifica, Inocybe infelix, I. infida, I. lateraria, I. sambucina, I. frumentacea, and I. repanda.

(D) Sanguinareus: causing gastro-intestinal symptoms followed by jaundice, anaemia and haemoglobinuria with low mortality. Species definitely incriminated—Helvella esculenta, but Morchella esculenta may contain a similar poison.

(E) Cerebralis: symptoms of transient excitement and hallucinations caused by only two species Panoeolus papiliomaceous and P. campanulatus.

Edible Fungi

There is no general infallible method of distinguishing edible from poisonous fungi. Several varieties or closely allied species in this country are edible and wholesome. The only safe procedure is to learn to identify certain well-recogized species by their botanical features, as the field mushroom, or the horse mushroom, and to avoid those growing under trees or in woods, as it is easy to make mistakes with the numerous varieties, some of which sport bright colours and are very poisonous. Even in the case of those known and correctly identified, caution must be exercised. It is essential that they should be fresh and be free from attacks by insects, or other organisms causing decomposition. Mushrooms are indigestible when eaten raw and unwholesome when decomposed.

Friese (1948) studied the keeping qualities of cooked and frozen edible fungi and noted the changes as measured by the *p*H, etc. He concluded that cooked or frozen fungi may be eaten with impunity before the reaction has become 'alkaline', but advises that fungi cooked with potatoes should never be warmed up.

Common Mushroom (*Psalliota campestris*)

The distinguishable features of the common mushroom are as follows: grows usually in short grass in open pastures, uplands or downs, in summer and autumn, and often growing in fairy rings in grass of a dark green shade. In the young or 'button' stage it is whitish and nearly round. Later the cap ($1\frac{1}{2}$ to 4 inches across, margin incurved) expands and becomes hemispherical and nearly flat. The mature cap is white or brownish-white in colour, skin dry, silky, smooth and peels easily and cleanly. The white stem which is $1\frac{1}{2}$ to 3 inches long and about $\frac{1}{2}$ to $\frac{3}{4}$ inch in diameter, is solid but

slightly pithy, enlarged below and requires a twisting movement to break it off. There is a membranous ring round the middle or towards the top; no sheath near top or at base. Flesh is white, thick and soft, colour changing to reddish or dirty brown when broken or cut; gills thin and crowded and not joined to stem. Spores egg-shaped and smooth. Colour whitish in 'button' stage, but becoming pink and finally dark purplish-brown to black. Odour earthy but not disagreeable. Taste somewhat earthy but pleasant.

Horse Mushroom (*Psalliota arvensis*)

An excellent description of this mushroom is given in Bulletin No. 23 (1935) Ministry of Agriculture and Fisheries, as follows:

> This species is larger than the common mushroom, usually 4 to 6 ins. across, though specimens up to 8 ins. across are not uncommon. The cap is at first almost globose, then hemispherical, and finally becomes almost flat. It is whitish in colour and silky-smooth, and becomes slightly stained with pale brownish-yellow when injured. The stem is white, sometimes stained with brownish-yellow, stout, thickened at the base, with a large spreading *double* ring towards the upper part. The gills are at first white, then finally dark reddish-brown. The flesh is firm, thick, white, and sometimes tinged with yellow.
>
> The horse mushroom is common in summer and autumn in pastures and beneath scattered trees, where it sometimes occurs in large rings, termed 'fairy rings'. It differs from the common mushroom not only in its larger size, but also in the flesh not becoming brown when cut and in the gills remaining dry when old.

Poisonous Fungi

The poisonous fungi found in this country are: Death Cap or Deadly Amanita (*Amanita phalloides*); Bulbous Agaric or False Death Cap (*Amanita mappa*); Fool's Mushroom (*Amanita verna*); Destroying Angel (*Amanita virosus*); *Entoloma lividum* (Leaden entoloma); Fly Agaric or Scarlet Fly Cap (*Amanita muscaria*); Warted Agaric or False Blusher (*Amanita pantherina*); Crested Lepiota (*Lepiota cristata*); Purple Agaric (*Cortinarius purpurascens*); Yellow-staining Mushroom (*Psalliota xanthoderma*); Verdigris Agaric (*Stropharia aeruginosa*); Red-staining Inocybe (*Inocybe patouillardi*); *Inocybe fastigiata.*

Of the above species the Death Cap and the Fly Agaric are the most frequent cause of poisoning.

Death Cap or Deadly Amanita (*Amanita phalloides*)

The death cap is said to be the cause of 90 per cent of the deaths caused by fungus poisoning. Ford (1909) calculated that 12

to 15 deaths occurred annually in the United States from this species alone. Dettrich (1924) estimated that in Germany it caused 80 to 90 deaths every year. It is extremely poisonous, very small quantities of the fungus causing intense suffering and sometimes death. Children are more susceptible than adults. Ford (1909) described the symptoms of poisoning by *Amanita phalloides* as follows:

Following the consumption of the fungi there is a period of six to fifteen hours during which no symptoms of poisoning are shown by the victims. This corresponds to the period of incubation of other intoxications or infections. The first sign of trouble is sudden pain of the greatest intensity localised in the abdomen, accompanied by vomiting, thirst, and choleraic diarrhoea with mucous and bloody stools. The latter symptom is by no means constant. The pain continues in paroxysms often so severe as to cause the peculiar Hippocratic facies, *la face vulteuse* of the French, and though sometimes ameliorated in character, it usually recurs with greater severity. The patients rapidly lose strength and flesh, their complexion assuming a peculiar yellow tone. After three to four days in children and six to eight in adults the victims sink into a profound coma from which they cannot be roused and death soon ends the fearful and useless tragedy. Convulsions rarely if ever occur and when present indicate, I am inclined to believe, a mixed intoxication, specimens of *Amanita muscaria* being eaten with the *phalloides*. The majority of individuals poisoned by the 'deadly Amanita' die, the mortality varying from 60 to 100 per cent. in various accidents, but recovery is not impossible when small amounts of the fungus are eaten, especially if the stomach be very promptly emptied, either naturally or artificially.

Ford (1923) collected 990 cases of poisoning from *Amanita phalloides* of which 381 had proved fatal.

The death cap is found in woods and adjoining pastures. Greenish or yellowish-olive, occasionally white, in colour, the cap (3 to 3½ inches across) is streaked with dark fibres and is sticky when moist. The stem is 3 to 5 inches in length and about ½ inch thick, whitish in colour but sometimes tinged with green and has a loose silky ring towards the upper part. The base is bulbous and is sheathed by a large yellowish-white cap which is more or less buried in the soil. The gills are white with sometimes a slight greenish tinge. The flesh is white with a greenish colour under the outer skin. When old, the fungus has a foetid odour.

The poisonous properties of *Amanita phalloides* were investigated originally by Letellier in 1826. He isolated a substance which he termed 'Amanatin'. Later, several other workers attempted to obtain the active poisonous principle of the fungus, and in 1891 Kobert extracted a powerful haemolytic poison (acting upon the red corpuscles of the blood, dissolving out the red colouring matter)

which he named 'phallin'. In 1901 the same worker demonstrated a poisonous substance in alcoholic extracts of *Amanita phalloides*, and after further experiments concluded that the active principle was an alkaloid.

Ford (1906) investigated these substances and found that phallin (which he termed 'Amanita haemolysin') lost its haemolytic property when heated to 70°C. or on exposure to weak acids or alkalis and by the action of pepsin or pancreatic juice. Nevertheless, the substance, after heating, still retained its toxicity for experimental animals and gave rise to lesions similar to those seen in human cases poisoned by the fungus.

Ford and Bronson considered that Amanita haemolysin was of little importance in cases of poisoning. They concluded that the extracted heat-resisting substance (named 'Amanita toxin') which was devoid of haemolytic properties could not be regarded as a protein or glucoside, but was the active principle responsible for the fatal human cases following the consumption of the fungus *Amanita phalloides*. It ranked as one of the most powerfully known poisons of plant origin.

Damon (1928) remarks:

> From our present knowledge of the subject the active principle in poisoning from the species of fungus undoubtedly appears to be the amanita toxin, with amanita haemolysin playing but a minor part, if any at all, in the intoxication.

According to Nobécourt, Martin, and Lipmann (1940) and Hoechstetter (1943) *Amanita phalloides* contains two toxins, one which is thermostable and resembles the action of phosphorus and the other a haemolysin which is thermolabile. Later, German chemists carried out detailed investigations into the Amanita toxin and isolated three toxic substances in crystalline form which they named amanitine, B-amanitine, and phalloidine.

Supplies of serum for the treatment of cases of poisoning from *Amanita phalloides* can now be obtained from the Public Health Service Laboratory, Colindale, London, and at various regional laboratories in England and Wales. It is recommended that the serum treatment be instituted as soon as possible after ingestion of the fungus, the average dose being 40 ml., administered either hypodermically or preferably intra-muscularly.

Rautavaara (1950) has recorded a useful survey dealing with the poisonous and supposedly poisonous fungi of Finland. Many of the fungi mentioned have a wide geographical distribution, making this review of general interest. *Amanita phalloides*, which

is responsible for most of the deaths due to fungus poisoning in Europe, is rare in Finland.

ILLUSTRATIVE OUTBREAKS

Plowright (1905) reported several typical cases of poisoning by *Amanita phalloides*. One, a boy of 12, ate a small portion of the raw fungus, at 11.30 a.m. About 1 a.m. the next morning (13 hours later) he commenced vomiting and suffered from thirst and diarrhoea. These subsided and he was able to eat his breakfast but soon afterwards the vomiting and diarrhoea returned. Later, however, his condition greatly improved. These periods of attack and remission were repeated until the fifth day when the boy had slight convulsions and died.

Plowright (1905) also records an interesting outbreak which occurred in a family of 4 persons, 2 of whom were severely poisoned by the fungus, but recovered, while the other 2 died.

A man, his wife, son, and daughter gathered and consumed 4 to 4½lb. of Amanita phalloides. The mother and son ate the fungus in a raw state and early the following morning were taken ill, as were the father and daughter later. The usual symptoms, i.e. thirst, sweating, vomiting, gastro-intestinal disturbance and intense abdominal pain were observed. The son developed convulsions, distortion of the face muscles, dilation of the pupils, involuntary oscillations of the eyeball, and he died 54 hours after eating the fungus. The mother developed jaundice on the third day and suffered from cramp-like pains. She aborted a 3 months' old foetus and on the fourth day was restless, with retracted head, almost unconscious, with complete anuria, respiration became irregular. She succumbed about 100 hours after ingestion of the fungus. The father exhibited similar symptoms, but on the eighth day felt better and eventually recovered. The daughter had diarrhoea with blood and mucous in the stools, great thirst, and enlargement of the liver. The diarrhoea gradually subsided and she slowly recovered.

Jackson (1946) records two cases, a mother and daughter who had eaten the poisonous fungi (*Amanita phalloides*) in mistake for mushrooms

The fungi had been picked in a field near Ipswich. They were ingested on a Saturday morning and by evening the mother had started to vomit. This continued all night and on Sunday morning a violent diarrhoea began with colicky abdominal pains and severe cramps in legs, feet and hands. By Monday, when she was admitted to hospital, she had

improved. A few days later she was constipated and had marked anorexia. She then had considerable tenderness beneath the right costal margin, but no other physical signs until she developed a typical erythema multiforme on trunk, arms and legs 8 days after eating the fungi, which cleared within 3 days. Her recovery was then uneventful. The little girl, aged 5, had eaten only a piece of one fungus and was not taken ill till the Sunday morning, when she was suddenly seized with severe diarrhoea and vomiting. By Monday she was delirious and in hospital, was noted as thin, but not notably dehydrated, 'parchmenty' in colour and texture of skin, and completely comatose. She was shocked and pulseless, but not cyanosed or jaundiced. Her respirations became more and more rare and gasping and she died that morning. Necropsy revealed a very intense uniform fatty degeneration of the liver.

Dubash and Teare (1946) record 4 fatal cases of poisoning from *Amanita phalloides* in England during 1944–5. They state:

In all four cases the main lesion appeared to be a severe toxic action on the liver and kidneys, leading to the rapid appearance of renal and hepatic failure. One striking feature was the constancy of the time factor between the ingestion of the poison, the onset of symptoms and death. The latent period before the appearance of symptoms of phallin poisoning was characteristically of 6 to 12 hours duration. The initial symptoms-complex of vomiting, diarrhoea and collapse was very constant. Gradually the clinical picture became dominated by signs and symptoms of liver and kidney damage. Other authors have described cases with marked jaundice and diarrhoea. Again, cases in which the picture was one of damage to the central nervous system with convulsions and generalised clonic spasms has been described—'forme pseudotetanique'.

Lewes (1948) records in detail poisoning due to *Amanita phalloides* in two German prisoners of war. Both patients recovered. He states:

The severity of the gastro-intestinal symptoms in the early stages of the illness contrasted with the mildness of the delayed hepato-renal damage. The delayed effects of poisoning were more severe in the patient with the longer silent period before the onset of symptoms and with the less pronounced and protracted diarrhoea and vomiting. The ability of the gastro-intestinal tract to eliminate the toxin of *Amanita phalloides*, and the thoroughness with which this elimination is assisted by early and efficient gastric and colonic lavage, are regarded as factors of the greatest importance in determining recovery.

Steyn, Steyn, Van Der Westhuizen, and Louwrens (1956) record an outbreak of mushroom poisoning caused by *Amanita phalloides* at Ermelo, Transvaal; 4 white adults, 1 Bantu adult, and 4 white children were affected. All but the 4 white adults died. The symptoms which appeared 12 hours after eating the fungi were nausea, persistent vomiting, severe abdominal pain, and choleraic

diarrhoea, dehydration, and cramps. During the next few days the liver became enlarged and tender, the conjunctiva icteric, and the urine dark in colour. Temperatures were subnormal and restlessness gave place to stupor and coma. Gross findings at autopsy were enlargement and extensive fatty degneration of the liver and haemorrhagic erosions on the gastric mucosa.

Fly Agaric or Scarlet Fly Cap (*Amanita muscaria*)

This fungus, which causes severe illness and sometimes death grows under birches and firs and in woods. The name 'fly agaric' is derived from the fact that a decoction of the fresh fungus was formerly used as a fly poison. It somewhat resembles the edible amanita, but can hardly be mistaken for the common field mushroom as it is a brilliantly-coloured decorative species and is one of the most beautiful of the agarincini. The expanded flat and sticky cap (4 to 7 inches across) is of a scarlet or orange-red colour and covered with thick irregular whitish warts. The stem is white or yellowish in colour and 4 to 7 inches high. The base is bulbous and is encircled by several concentric rings formed by the remains of the 'volva' (a cup or sheath-like structure at the base of the stem).

Ford (1909) records a case (Count de Vecchi in Washington, D.C., in 1897) where this fungus (*Amanita muscaria*) was mistaken for the European variety of 'royal Amanita' (*A. caesaria* or *aurantiaca*) with fatal results:

The Count, an attaché of the Italian legation, a cultivated gentleman of nearly sixty years of age, considered something of an expert upon mycology, purchased, near one of the markets in Washington, a quantity of fungi recognised by him as an edible mushroom. The plants were collected in Virginia about seven miles fron the city of Washington. The following Sunday morning the Count and his physician, a warm and personal friend, breakfasted together upon these mushrooms, commenting upon their agreeable and even delicious flavour. Breakfast was concluded at half after eight, and within fifteen minutes the Count felt symptoms of serious illness. So rapid was the onset that by nine o'clock he was found prostrate on his bed, oppressed by the sense of impending doom. He rapidly developed blindness, trismus, difficulty in swallowing, and shortly lost consciousness. Terrific convulsions then supervened, so violent in character as to break the bed upon which he was placed. Despite rigorous treatment and the administration of morphine and atropine, the Count never recovered consciousness and died on the day following the accident. The Count's physician on returning to his office, was also attacked, dizziness and ocular symptoms warning him of the nature of the trouble. Energetic treatment with apomorphine and atropine was at once instituted by his colleagues, and for a period of five hours he lay in a state of coma with occasional periods of lucidity. The

grave symptoms were ameliorated and recovery set in somewhere near seven o'clock in the evening. His convalescence was uneventful, his restoration to health complete, and he is, I believe, still living. In this instance the Count probably identified the fungi as *caesaria* or *aurantiaca*. From the symptoms and termination the species eaten must have been muscaria.

Amanita muscaria contains the alkaloidal substance 'muscarine' which was isolated by Schmiedeberg and Koppe in 1869 and afterwards by other investigators. It has since been obtained in a crystalline form as the hydrochloride and in its action somewhat resembles pilocarpine. Patterson and Charles (1915) suggested, however, that there were probably other poisons present besides muscarine, because atropine, which was a perfect antidote for muscarine, did not entirely neutralize the effect of injections or decoctions of this species of fungus.

Savage (1920) remarks:

> Although muscarine is a powerful poison the symptoms it produces in the human subject are not identical with those produced by this type of mushroom poisoning. Also an infusion of the fresh fungus is very poisonous to flies while muscarine itself is harmless to those insects. While, therefore, it is reasonable to assume that muscarine plays a large part in the toxicity of this mushroom, it is probably associated with other poisonous bodies which have not yet been isolated and studied.

The characteristic symptoms vary considerably in intensity in individual cases, following the ingestion of this fungus. They usually appear in from 1 to 6 hours but shorter incubation periods have been recorded. There is salivation, sweating, lacrimation, giddiness, vomiting, and diarrhoea. Respiration is accelerated but the pulse is slower and irregular.

In most cases the pupils of the eyes are contracted and do not react to light and accommodation.

In severe poisoning, nervous and mental disturbances occur, and there is violent gastro-intestinal reaction and later delirium, convulsions, and sometimes death from respiratory paralysis.

Bulbous Agaric (*Amanita mappa*)

This is found frequently in woods from August to November. The species somewhat resembles the poisonous *Amanita phalloides*, with which it may be easily confused, and its odour is very unpleasant. Two or three inches across, the broad convex cap is whitish-yellow to brown in colour, and is flecked with brownish-yellow fragments of the ruptured volva (membrane sheath near

base of stem). The crowded narrow white gills often have a yellow edge. The tall, slender, round, white hollow stem, 2–4 inches in height, has a bulbous base. Volva is smoky-yellow in colour and the upper friable portion disappears, leaving a short thick margin free from stem and is separated from it by a groove.

Fool's Mushroom (*Amanita verna*)

This fungus, which is closely allied to the Death Cap (*Amanita phalloides*) and very poisonous, is comparatively rare in this country. It is characteristically white in colour and grows in damp situations in woods, especially under beech trees during the summer and early autumn. The conical white cap when expanded, is thin and slimy with a slightly brownish tint in the centre, the latter being somewhat depressed. Gills are white and broad. The stem with ring near top is long (about 4 to 5 inches) and slender. Volva at base is thick and sheathlike with a somewhat lobed margin. The flesh when broken has an unpleasant odour.

Warted Agaric (*Amanita pantherina*)

A very poisonous species is found in woods and pastures and on heaths during July to October. The taste and smell are unpleasant. The depressed convex, viscid, fleshy cap, which has grooves near the edges, is brownish-grey in colour and sprinkled (warts) in the depressed portion of the fungus with small pieces of the volva. The gills and flesh are white; the flesh does not change colour when cut. The attenuated round white stem has a bulbous base surrounded by a thin membranous concentric volva.

Purple Agaric (*Cortinarius purpurascens*)

This fungus is fairly common in woods from September to November and is found singly or in groups. The convex glutinous spotted fleshy cap is a purple-crimson to brownish-olive colour, and is peculiar in shape, having depressions and raised violet zones near its edges. The crowded broad gills are at first a bluish or purple colour, later, turning cinnamon or rusty-brown. The flesh is azure blue. The fibrous solid pallid azure blue stem has a bulbous base and somewhat marginate.

Yellow-Staining Mushroom (*Psalliota xanthoderma*)

Care is needed to distinguish this species from the edible horse mushroom which it somewhat resembles. It is found in woods,

pastures and hedgerows in the summer and early autumn. The taste and smell of the yellow staining mushroom is strong, foetid and unpleasant. The silky skin on the globular expanded fleshy cap (2 to 3 inches in diameter) is white, but if bruised or scratched (especially if moist) develops a bright yellow stain. This staining effect also applies to the lower part of the elongated fibrous, fleshy, smooth white stem which has a bulbous base. It turns yellow if cut. Gradually the white gills become pink and finally violet or brown in colour.

Verdigris Agaric (*Stropharia aeruginosa*)

This fungus is commonly found amongst grass and bracken in damp woods and pastures during the summer and early autumn. The bell-shaped cap, which is about 2 to 3 inches across, is at first greenish (or bluish-verdigris) in colour, especially in the young fungus, but later turns yellow. The flesh has a bluish tinge. The fairly long slender greenish-coloured fleshy stem, 1 to 4 inches, is covered (below the membranous ring) with small temporary scales. The gills which are attached to the stem are at first white, but later turn dark purple.

Red-Staining Inocybe (*Inocybe patouillardi*)

This fungus grows among grasses in woods and parks during the summer and autumn. It is at first creamy white in colour but gradually turns pinkish yellow and finally red. The conical or bell-shaped cap, about 1 to 3 inches in diameter, is covered with silky fibres. Later, however, it flattens out and the margin becomes lobed and torn. The white to straw-coloured stout stem, which is usually 1 to 3 inches long and about $\frac{1}{2}$ to $\frac{3}{4}$ inch thick, becomes streaked with red and eventually assumes this colour throughout. The stem is swollen near its base. The thick gills, which are fairly crowded, are whitish-pink in colour, turning to olive-yellow, later becoming blotched with red with a whitish edge. The flesh is thick, firm, and white, but stained red in parts and has a strong fruity smell. The spores, which vary in size, are bean-shaped, smooth, and brown in colour. The fungus when broken stains the fingers red. If eaten it causes giddiness, vomiting, and sweating. Several instances of illness, and even death, have been recorded following its consumption. There are several other species of Inocybe, all of which are probably poisonous.

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Chapter XIV

POISONOUS FISH AND SHELL-FISH

This type of food poisoning is not very common in this country, but there are many kinds of fish, especially those found in tropical waters, which if eaten, even when in an apparently healthy condition, sometimes produce symptoms of poisoning—symptoms which are more likely to occur if certain parts are consumed, such as the liver, roe, head, or intestines. Fish may become poisonous after feeding upon certain medusae and coral.

Fish are covered normally with a layer of a mucous substance (slime) which is composed of nitrogenous substances. Decomposition of fish is caused partly by certain non-living substances, namely enzymes which occur in small quantities in the flesh, and partly by bacteria which attack the flesh from the exterior surface as soon as the fish is dead. Bacteria are also present on or near the gills and in the gut. Research has proved, however, that there is no evidence that food-poisoning organisms are members of the normal flora of fish or that newly caught fish suffer ordinarily from salmonella infections.

Shewan (1949) points out that

> although the flesh and body fluids of new-caught fish are sterile, the external surface (slime and gills) and the gut, when food is present, can carry considerable bacteria loads. . . . In dead fish, the slime certainly acts as a good medium for bacterial growth, and it seems probable that infection in the living fish is kept down by continuous secretions and sloughing of the slime.

Fellers (1926), in a study of raw salmon spoilage, found numerous organisms in the mouth, gills, and slime of live salmon. He observed that bacteria penetrated the flesh under average conditions in from 24 to 60 hours, depending upon such factors as the size and species, temperature and methods of handling. The number of bacteria in the flesh increase rapidly with each 24 hours.

Symptoms of Poisoning

These are usually of two kinds: gastro-intestinal irritation with rapid prostration and sometimes urticaria; severe nervousness and convulsions.

In some fish a poisonous substance appears to be secreted at certain times of the year. For instance, the roes of pike, sturgeon, carp, bream, and turbot produce violent intestinal disturbance if eaten during the breeding season.

Abraham (1906) reported 28 cases of poisoning from the ingestion of infected pike. The symptoms were like those observed in typhoid fever, but examination for ptomaines and poisonous metals was negative. An organism of the *aertrycke* type, however, was isolated.

Jordan (1931) remarks:

> The season of the year at which the fish is taken is undoubtedly a factor of importance, and there is evidence connecting the presence of toxic constituents with the state of the reproductive organs. The ovaries of the sea urchin, which is eaten by some of the Mediterranean people, are said to be poisonous during the spawning season.

Fish roe poisoning is common in Russia and is a cause of severe gastro-enteritis. The sturgeon caught in Russian waters is salted and smoked for consumption, but this fish is often eaten in the raw state and numerous cases of poisoning of a toxigenic nature have been recorded. (Zlatogoroff and Soloview, 1927, and Siebera Schoumow 1894–5). Burova, Nasledisheva, Kats, and Denisova-(1935) and Dobrowsky (1935) found that *Cl. botulinum* occurs in the intestines of 10–12 per cent of the live Caspian sturgeons examined by them.

Certain varieties of fish are perfectly harmless if eaten as soon as they are caught, but become toxic if allowed to remain uncooked even for an hour. Flat fish maintain their freshness better than round fish. Those of an oily nature tend to decompose rapidly. Of the fish ordinarily consumed in this country, mackerel has the worst reputation for occasionally causing illness, possibly due to the rapidity of decomposition; this fish should be eaten as soon as possible after being caught. Shewan (1949) remarks:

> Incidentally, only in two groups of fish—*Scombridae* (mackerel, tunny, etc.) and *Clupeidae* (sardines)—is there definite evidence of harmful products causing food poisoning being produced during the spoilage process by the normal bacterial flora, i.e. not by the recognised food poisoning pathogens. It has been found that the flesh of newly-caught fish of these groups contain histidine which is readily broken down by the normal bacterial flora to histamine. This later compound is one to which many people, but not all, are very sensitive causing nausea, diarrhoea and sickness.

Fresh mackerel have a greenish-blue colour and the markings are distinct. When stale, a dark reddish colour appears round the

gills and the flesh 'pits' easily. This is one of those instances where the condition known as 'rigor mortis' is a guide; decomposition proceeds rapidly. There is no doubt that of all the protein-rich foods, fish are normally among the least likely to cause food-borne infections. Spoilage organisms usually cause fish to become unpalatable before any pathogenic organisms can develop their toxins.

Geiger (1955), as a result of his investigations and experiments on the rôle of histamine in poisoning with spoiled fish, is of opinion that histamine is present only in traces in fresh fish, such as mackerel, sardines, tuna, and salmon, but the amount increases rapidly during spoilage up to values of 120 mg. per 100 gm. fresh meat within 24 hours. This happens with the round fishes, but in the flat fishes (sole, halibut, etc.) only small amounts are produced during spoilage. It has been suggested by a number of authors that food poisoning from spoiled fish may be due to histamine.

Geiger's experiments with tuna inoculated with an unidentified bacterial strain at 37°C. for 56 hours, produced large amounts of histamine, i.e. 190–210 mg. per 100 gm. of fish. This was fed to dogs, cats, rats, mice, and guinea pigs, but caused no symptoms of poisoning. He concludes that toxic histamine played at most a very minor rôle.

According to Günther (1880) the flesh of certain members of the herring family, such as *Clupea thryssa* and *Clupea venenosa*, are poisonous. The former—all parts of which are poisonous—has been known to cause death before being actually swallowed.

Appearance and Characteristics of Fresh Fish

A freshly caught fish is moist, smooth, glistening, and has an attractive appearance. The smell is not unpleasant. Rigidity and stiffness are sure signs of freshness. Scales are bright and not easily detached from the skin. After being packed in ice, fish may appear pitted all over, but they are quite fit for human food. The eyes are prominent, bright, full pupils jet black in colour and the corneas transparent; gills firm, clean (free from slime) and usually of a neutral red colour, but the tint varies with the species. The flesh is firm and elastic to the touch, well attached to the bones, and not discoloured along the backbone. That around the kidneys and caudal vein is light in colour. There is no discoloration of the abdominal walls and the blood in the visceral cavity and under the sound is bright red.

Cured Fish

Kippers and bloaters of good quality are bright and glossy in appearance and free from smuts and dirt. The smell is pleasant. Kippers are golden brown in colour, but if dyed, more reddish-brown especially on the fins. The skin and cut surfaces are oily, but the latter should not have a rancid taste or smell. The flesh is firm, not torn or gaping, and there is no discoloration of the belly walls. Bones should not crackle on pressure. Red herrings are crisp and dry and without an offensive smell. Generally speaking, smoked fish should feel firm and springy, not flabby, sticky, or moist. In the case of finnan haddock or smoked fish fillets, the flesh is firm and has a straw-yellow colour.

The standard required by the Torry Research Station (1952) for kippered herrings is:

> Skin should have an attractive surface sheen. Scales on skin intact. Fish clean and have no offal or gut sticking to it. Belly not slimy or dark green in colour. Belly walls sound—a small hole or tear shows the fish to have been left too long before gutting. No white deposit particularly on gills. The fish should not be sticky. Colour matter of preference; dyeing is now universal.

Signs of Staleness and Commencing Decomposition

The fish loses its rigidity and becomes soft and limp. Odour sour and disagreeable, later becoming offensive. The scales lose their sheen, are dry and dull looking and easily detached from the skin. Eyes appear dull and shrunken in the sockets, pupils milky-white or greyish. The gills are dirty red, brown or greyish in colour, later becoming greenish and slimy. The flesh is soft and flabby, easily pits on pressure (except hake) and is dark red or brownish in colour, especially along the backbone. It is easily stripped from the bones. The degree of discoloration, however, depends upon the time elapsing since the fish was caught. Walls of abdominal cavity are soft, pulpy, discoloured, sometimes showing a jelly-like appearance, and emit an unpleasant or putrid odour. The blood under the sound is dark red or chocolate coloured and has an offensive smell. The kidneys, being diffuse and friable organs, rapidly decompose and change colour from a light to a dark brownish jelly. Although fish may have been well iced, they soon deteriorate and become stale. Newly spawned or diseased fish soon commence to decompose. Roughly handled, battered, bruised, or carelessly packed fish do not keep well.

Cured fish become dull looking, the flesh is soft and has a stale smell, especially under the backbone.

Finnan haddock: the flesh is wet, soft, and clammy and there is a reddish discoloration along the backbone.

Smoked fish fillets: the flesh is tinged yellow or pink, breaks down easily, and has an offensive smell.

Flat fish: the abdominal cavity and the surrounding flesh is discoloured.

Shewan (1949) remarks:

The onset and revolution of *rigor* varies somewhat with species (Schlie 1934) with the degree of rough handling received and with temperature (Anderson, 1907). For commoner commercial species of 'white' fish stowed in ice, the revolution of *rigor* lies between $1\frac{1}{2}$ and $2\frac{1}{2}$ days—cod and haddock emerging sooner than the flat fishes. Whitings which are notoriously poor 'keepers' appear to be an exception, *rigor* resolving in about 1 day.

Rapid Inspection of Fish

Some investigations and experiments were carried out at the Torry Research Station, Aberdeen, to find a simple and speedy test of the freshness of fish that would be suitable for use during their inspection in the ordinary markets. The results indicated that certain colourless tetrazolium compounds might be useful in the assessment of fish quality, since they are reduced by bacterial dehydrogenases to red-coloured formozans. The depth of the colour and the speed of its development are functions of the total bacterial activity.

In the experiments, the test papers were impregnated with the tetrazolium salt and laid on the wet surfaces of the fish which were at various stages of spoilage in ice. The depth of the colour produced within a fixed time was determined against standard papers or by eluting the coloured compound and estimating it colorimetrically. Sensory assessments of quality were also made.

It was found that very fresh fish, i.e. fish kept for not more than a week in ice, produced no colour, while very stale to putrid fish quickly caused the test papers to turn purplish red. These tests were made with cod and haddock caught off Aberdeen.

Kleeman, Frant, and Abrahamson (1941), in recording two outbreaks of food poisoning, traced to smoked white fish, butter-fish and carp, made the following recommendations:

Since lightly smoked and lightly salted smoked fish is perishable, and readily serves as a medium for the growth of pathogenic organisms, it

should be treated as such a type of food product, and the carrying out of the following precautions is indicated:

1. The building in which the food processing takes place should be of sanitary construction throughout, with walls, floors and ceilings made rat-proof and readily kept clean.

2. There should be facilities for sterilisation of all utensils and equipment which come in contact with the products, and the cleaning and gutting of fish should be done under a constant and ample flow of potable water.

3. There should be an adequate and suitable sewage disposal system and sanitary plumbing to protect the products. Live stock and other animals should not be permitted on or near the premises.

4. All workers engaged in the processing phases of this industry should have regular medical examinations, including laboratory tests when such are indicated. Cleansing of the hands, especially after visiting the toilet, cannot be over-stressed.

5. Only salt which has been redissolved and purified after an initial mining or crystallisation should be used in making up the brine solutions for processing these products.

6. The brining process should be carried out under adequate refrigeration, that is, at a temperature of 50°F. or below.

7. Preliminary drying of the fish prior to smoking should be accomplished as quickly as possible with the aid of rapid circulation of warmed air.

8. Adequate refrigeration of 50°F. or below should be provided at all subsequent stages of storage, transportation, distribution, and retail display.

9. Efforts should be made to educate the consumer with regard to the perishable nature and disease hazards of this type of food.

Shewan (1949) says:

As all types of processed fish have been incriminated from time to time in food poisoning, it might be of interest to consider finally the effect of the various processing techniques on the three main groups of food poisoning pathogens, viz.: the Salmonella, Cl. Botulinum and Staphylococci.

The relevent data are given in the accompanying table.

Quick Freezing and Glazing

In recent years quick freezing, glazing, and cold storage afford almost perfect preservation of fresh and lightly cured fish of all kinds for a period of months. Fish treated in this manner remain palatable and in good condition.

Reay, Banks, and Cutting (1950) stress the following:

The period elapsing between the removal of the fish from storage in ice to the commencement of freezing should be kept as short as possible. The 'critical' rate of freezing recommended for fish (from 32° to 23°F. (0° to —5°C.) in 2 hours) is one which all modern types of 'rapid'

THE EFFECT OF PROCESSING ON FOOD POISONING PATHOGENS
(after Shewan, 1949)

Type of Process	*Type of Micro-organism*					
	Salmonella. SPP.	*Cl. Botulinum*			*Staphylococci*	
	Vegetative form	*Vegetative form*	*Spores*	*Toxin*	*Vegetative form*	*Toxin*
Chilling (down to 0°C.)	Growth occurs at 10°C., but none below 4°C.	No growth at 5°C. after 108 days. Growth at 10°C. in 27 to 98 days.	No growth of detoxified spores at 5° or 10°C. after 47 days. Growth within 13–22 days at 15°C.	No effect.	No growth or toxin formation below 4°C. Growth and toxin formation in 3 days at 18°C. and 12 hours at 37°C.	No effect after 2 months at — 4°C.
Freezing (0°C. and below)	May survive 14 months at)—18°C.	May survive at —20°C.	Survives at —16°C. for 14 months.	Not destroyed after 14 months at —16°C. or 2 months at —79°C.	Survives 14 months at 18°C.	No data.
Drying and Dehydration	No growth if less than 30 per cent H_2O present.	No data.	Probably survive.	Not destroyed 6 months after drying.	No growth if less than 20–30 per cent H_2O present.	No data.
Smoking: (*a*) Hot Smoking	Do not survive careful smoking.	Probably killed.	Probably survive.	May be partially detoxified.	Do not survive careful smoking.	No data.
(*b*) Cold Smoking	Probably not sufficient to destroy.	No data.	Probably survive.	May be partially detoxified.	Killed after $2\frac{1}{2}$ hours.	No data.
Salting	No growth above $6\frac{1}{2}$ per cent NaCl. Can survive 22 days in salt fish and 94 days in salt brine (at 5°C.)	Survives in 10–12 per cent salt. Grows only in heavy inoculation in 8 per cent salt.	Survive long periods.	No data.	Can survive and grow even in concentrated salt.	Can be produced in salt fish.
Canning	Killed.	Killed.	Killed.	Destroyed $1\frac{1}{2}$–5 minutes at 80°C.	Killed.	Relatively heat stable. Long boiling 60 min. or auto-claving 20 min. 15–16 lb. pressure, gradually decreases potency.

freezer should accomplish with all classes of unwrapped fish or of closely wrapped packages of fish up to a thickness approaching $3\frac{1}{2}$ inches. 'Overall' freezing at this rate will occupy some $3\frac{1}{2}$ to 4 hours, assuming that the fish enter the freezer at 40° to 50°F. (4° to 10°C. and leave it at 0° to 10°F. (18° to —12°C.). In order to secure proper thermal condition and contact in the pack, fillets should be packed tightly together in blocks of regular size and shape.

In glazing, the frozen fish are dipped in, or sprayed with water, the temperature of which should be well below 32°F., thus reducing the rise in temperature of the fish as the adhering water freezes the glaze. Herrings must be rapidly frozen whilst in a fresh condition, well glazed and stored at temperatures below 0°F. (—18°C.). They can be stored in practically unchanged condition for 3 months at —5°F. (—21°C.) or for 6 months at —20°F. (—29°C.). At these low temperatures deterioration proceeds very slowly and even after 9 months at —20°F. (—29°C.), the fish are still in good condition. [Food Science Abstracts, 1950.]

It is important to remember that freezing does not materially reduce the bacterial load on fish; nor does it destroy any toxins present. Thus contaminated fish will still be in the same condition after quick freezing and cold storage.

Poisonous Tropical Fish (Ichthyosarcotoxism)

Poisonous fish are widely distributed throughout all warm seas but are particularly numerous around certain island areas in the Caribbean, Central and South Pacific Oceans.

More than 300 species have been reported from time to time as causing fish poisoning outbreaks. Among those well known are the different varieties of wrasse, certain species of barracuda found in West Indian waters, the parrot fishes, so named from their brilliant colouring; the toad fishes, file fishes, moray eels, trigger fishes, and the Tetrodontidae family (globe, puffers, and balloon fishes) comprising many important species, which are widely distributed along the coasts of Japan, China, East Indies, and Africa. The most poisonous fish are the Tetrodonchrysops, *perdalis*, *vermicularis*, and *poecilonotus*, while the less poisonous are the Tetrodon *rubripes*, *porphyreus*, *stictonotus*, and *rivulatus*. The Japanese chemists gave the name Fuga-poison (fuguismus) to that found in the ovaries and testes of the various species of Tetrodon, which causes so many deaths among this race. The consumption of one roe causes serious illness in a few moments, which may terminate fatally. It is believed that the same poison exists in all the fishes named. Poisoning by their consumption is acute and the onset of symptoms rapid.

Tjakalong, a variety of 'bomto' (*Euthynnus pelamis* L., more probably 'skipjack', *Katsuwonus pelamis*), a fish inhabiting the sea near Indonesia, is reported to become toxic (when exposed to the tropical heat) without showing any perceptible signs of spoilage. This condition is due to certain bacteria which produce histamine.

Von Bonde (1953) points out that recent research has shown that the South African blaasop or toby does not differ greatly from the American puffers or the Japanese fuga.

Wynter Blyth (1920) describes a typical case of poisoning by Tetrodon:

> A man in Kitshin (Japan) at 2 p.m., ate five pieces of a tetrodon (species not known). Four hours afterwards he complained of an uneasy feeling in the epigastrium the pulse at that time was normal. Vomiting was excited by tickling the back of the throat. Quite suddenly the patient was incapable of walking and was soon completely paralysed. Motion of the tongue was difficult and his speech indistinct. Later, cyanosis, diminished frequency of breathing and dilation of the pupil were observed. The corneal reflex disappeared and the body temperature sank. Artificial respiration and injection of camphor and strychnine gave no relief and death quickly followed five hours after the meal.

The nature of this poison has been studied by Takahashi and Inoko (1890), Micera and Takeski (1890), Tahara (1911) and other observers.

Paetro (1956) records four outbreaks of fish poisoning after consumption of great barracuda, all from fish caught off the coast of Florida, the first occurring in May 1954 when 5 persons were ill within 1 to 2 hours after eating the fish. Three cats fed on barracuda died within 24 hours. In the second outbreak, in June, 7 out of 10 people, after a dinner of barracuda, became ill in about 4 hours. The third was in July and involved 9 persons and all who ate the barracuda were ill in 1 to 5 hours. The last outbreak occurred in August and involved one family of 3, all of whom were ill after 7 to 10 hours. The symptoms were similar in all the outbreaks and included nausea, diarrhoea, numbness, and other sensory defects as loss of speech and partial loss of touch. The symptoms in some cases lasted up to 5 weeks.

According to Norman (1931),

> There are a number of fishes which, although without definite poisonous organs, have their flesh more or less permeated with poisonous substances, taking the form of alkaloids of a particular kind called leucomaines. This may be regarded as a special form of protection saving the species by poisoning its enemies. To eat certain species such as muki-muki, or death fish of Hawaii, is to invite certain death.

Strom and Lindberg (1945) record an outbreak of poisoning following the consumption of tunny fish (*Thynnus thynnus*). Three persons were affected. Ten minutes after eating the fish, flushing of the skin appeared with intense headache. Palpitations and rigors followed and the temperature became sub-normal. The severe symptoms lasted from one to five hours. Slight headache persisted until the following day when recovery was complete. Two further outbreaks are described with similar symptoms in 3 or 4 subjects respectively after eating fish cakes and fried tunny. A saline extract from the fish which caused the first outbreak produced erythema and urticaria when injected intradermally in a dilution of 1 in 100,000.

The active substance was soluble in water and alcohol, thermostable and resistant to desiccation, and gave negative reactions to tests for proteins. The Zimmermann test for histamine was positive. Injection of the extract into guinea-pigs caused death with spasm of the bronchial muscles, and the extract produced contraction of the isolated guinea-pig uterus. On the basis of these tests, the active substance in the fish was considered to be histamine, perhaps produced by the decarboxylation by enzymes of histidine present in the fish protein.

Regarding the nature of ichthyosarcotoxins, Halstead and Lively (1954) are of the following opinion:

> The chemical and pharmacologic properties of most ichthyosarcotoxins, fish poisons, are unknown. Tetraodontoxin or puffer poison has been studied to some extent by the Japanese. Puffer poison, in its purified state, is a white hygroscopic powder, readily soluble in water and insoluble in the ordinary organic solvents. Tetraodontoxin has been assigned the provisional chemical formula of $C_{16}H_{31}NO_{16}$. Japanese scientists are of the opinion that tetraodontoxin is neither a protein, an alkaloid, nor a protamine. The exact chemical structure and source of the poison are still unknown. Moreover, it is not known whether the ichthyosarcotoxins that are found in such fishes as the snapper, grouper, and moray eel are related to puffer poison, or whether they are a different compound. Most fish poisons (exclusive of puffer poison) appear to have a composite physiologic action on humans. Many of the symptoms are similar in nature to those produced by such compounds as aconite, muscarine and curare. Whether or not ordinary fish toxins are true alkaloids remains to be seen. These fish toxins are water soluble and relatively heat stable. Ordinary cooking procedures do not destroy or appreciably alter the virulence of the poison. The state of freshness of the fish has no bearing on the production nor the virulence of the toxin because putrefaction is not a contributing factor in this disease.

These observers divide clinical fish poisoning into four types: Tetraodon (puffer) poisoning; Gymnothorax (moray eel) poisoning;

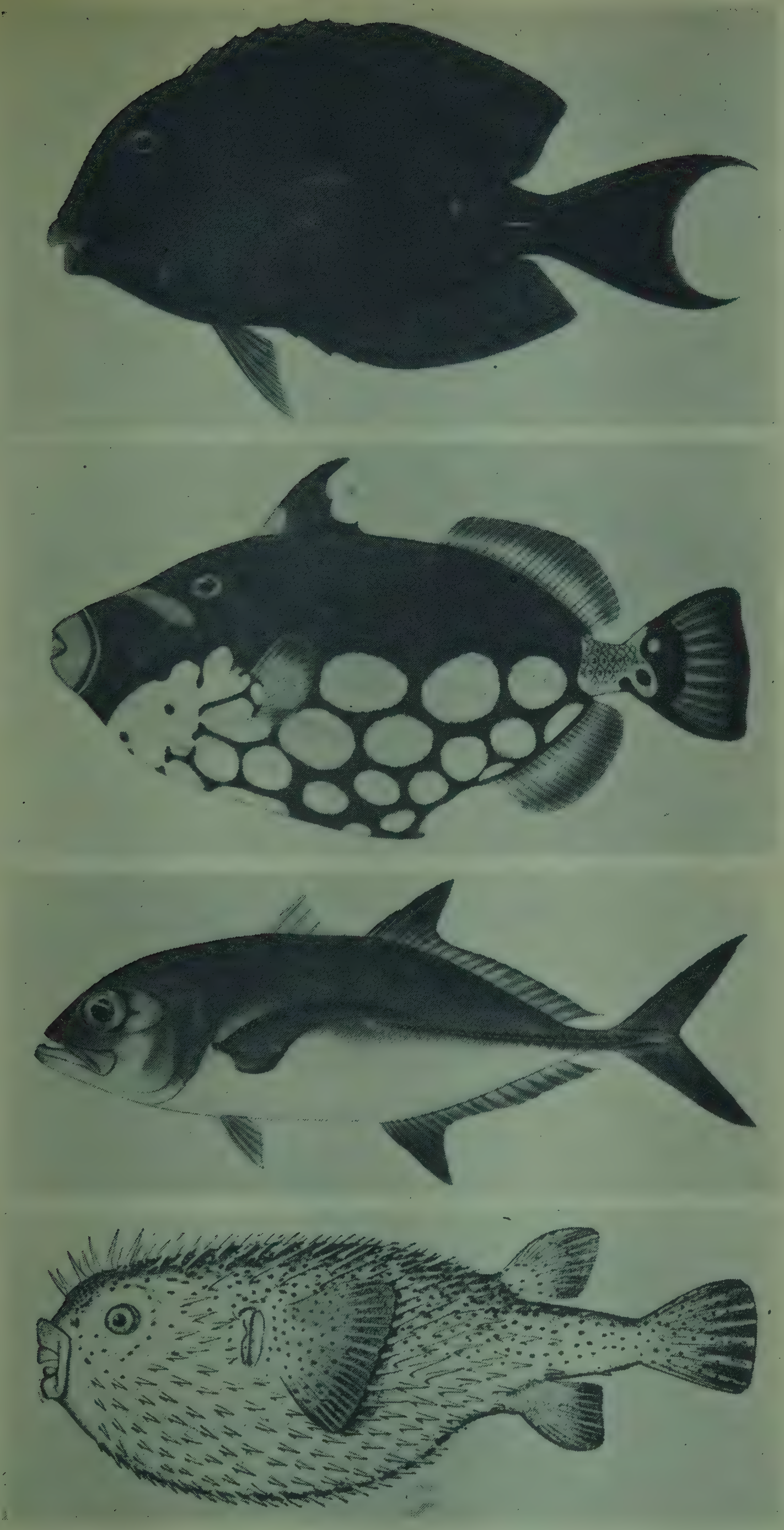


PLATE 25 (reading from top down). Surgeonfish (*Ctenochaetus strigosus*). Triggerfish (*Balistoides niger*). Pompano (*Caranx sexfasciatus*). Porcupine fish (*Diodon hystrix*)

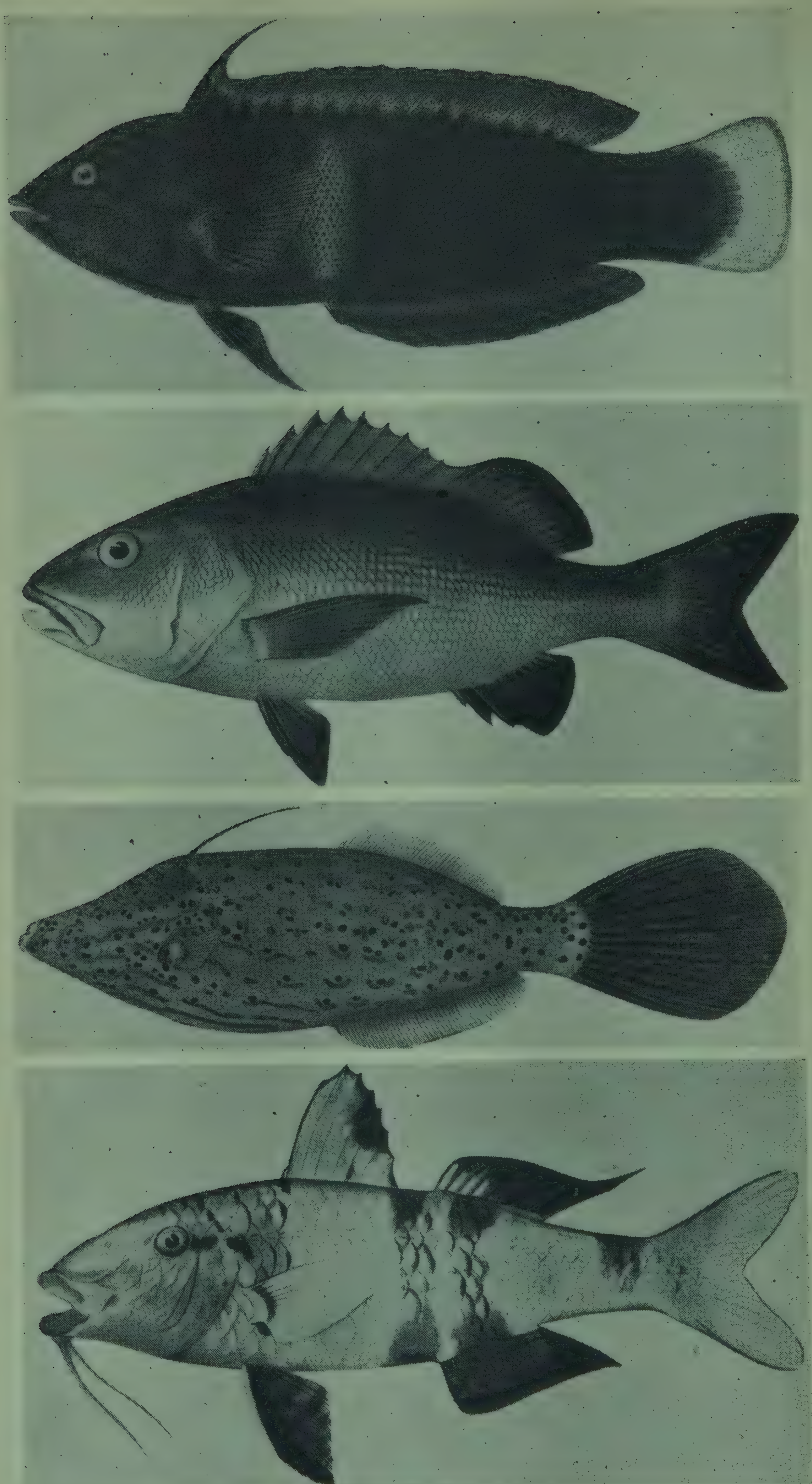


PLATE 26 (reading from top down). Wrasse (*Coris gaimardi*). Red snapper (*Lutjanus vaigiensis*). Filefish (*Alutera scripta*). Surmullet, or goatfish (*Parupeneus trifasciatus*)

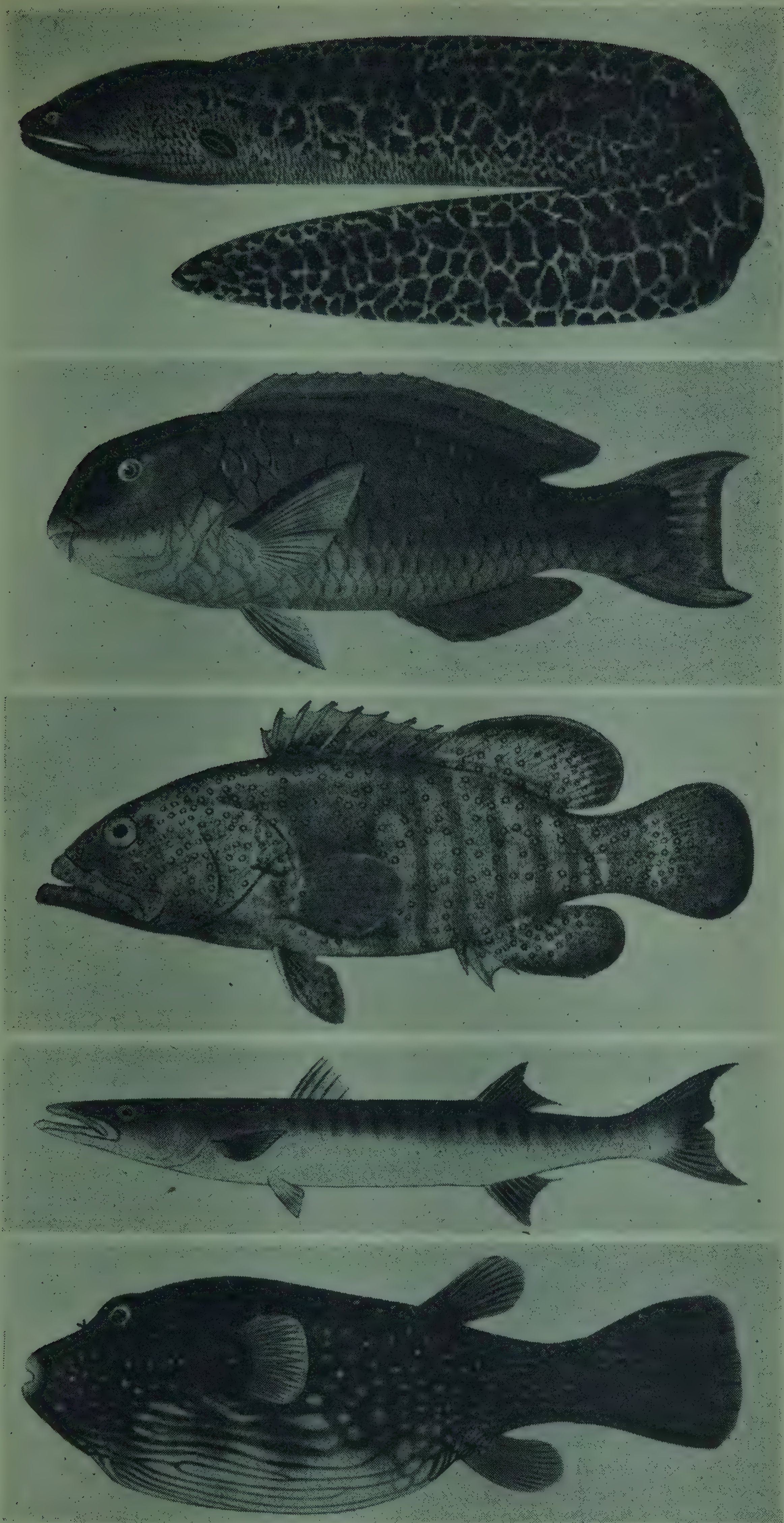


PLATE 27 (reading from top down). Moray eel (*Gymnothorax javanicus*). Parrot fish (*Scarus microrhinus*). Sea bass, or grouper (*Cephalopholis argus*). Barracuda (*Sphyraena barracuda*). Puffer (*Arothron hispidus*)

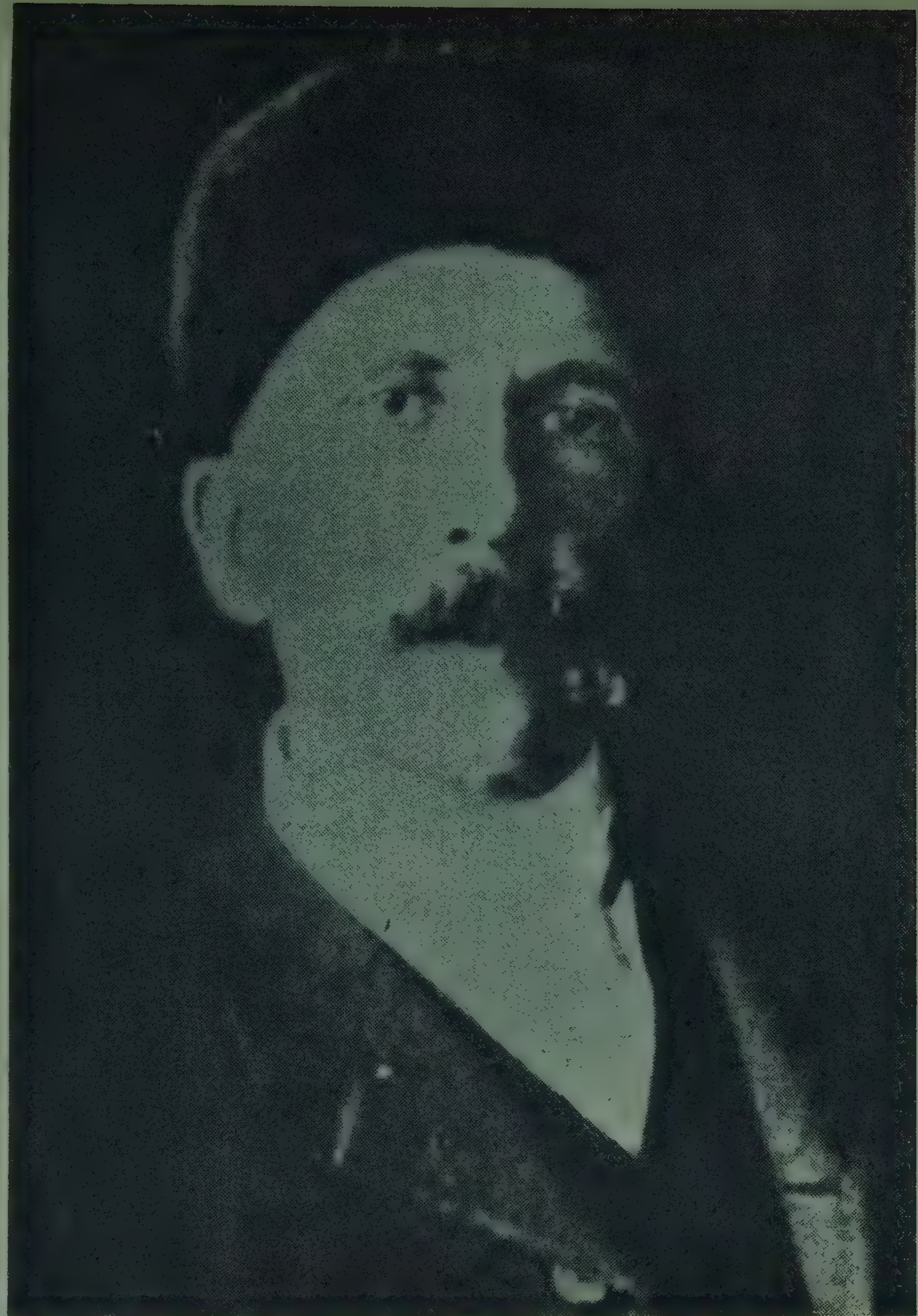
(*a*) R. W. Dodgson, M.D.

(*b*) Mussel purification: hosing the mussels

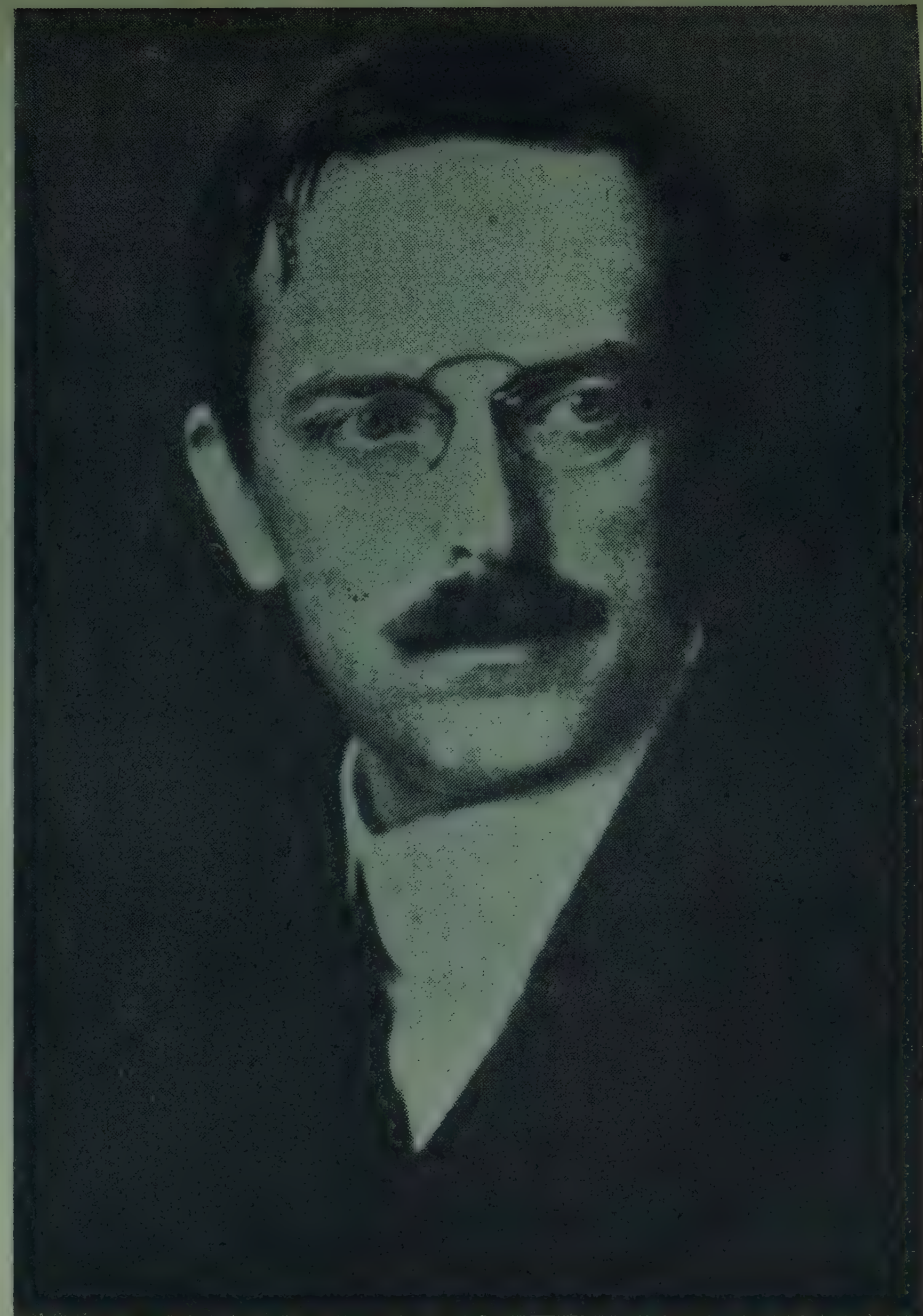
PLATE 28

PLATE 29. Bagging purified mussels

PLATE 30. Mussel purification tanks



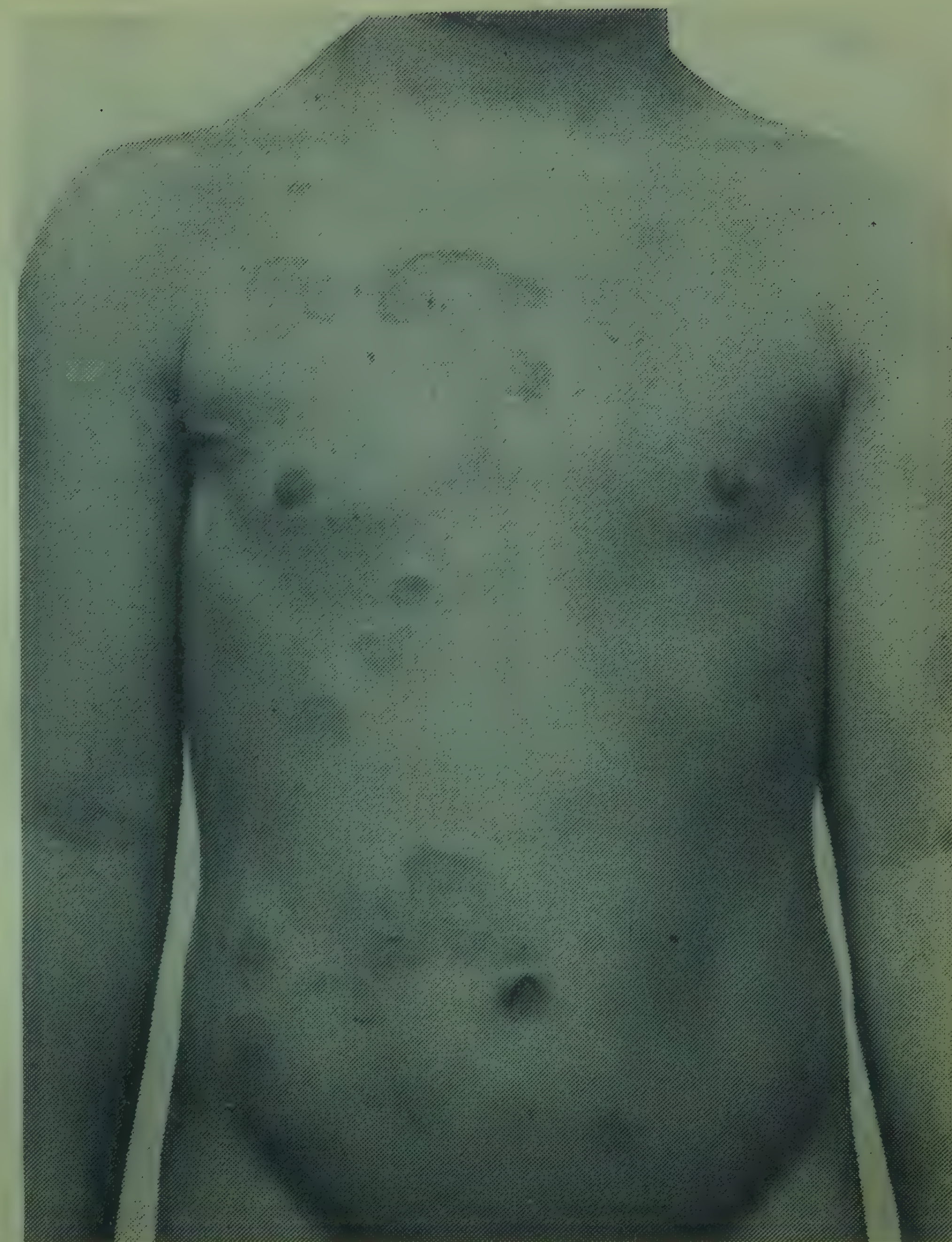
(*a*) Charles Richet



(*b*) Clemens von Pirquet

PLATE 31

(*a*) Allergic reaction to eggs



(*b*) Urticarial rash

PLATE 32

Ciguatera fish poisoning (exclusive of puffer poisoning) resulting from the ingestion of fishes in the Caribbean area (and the term Ciguatera is recommended to be used to designate the milder form of ichthyosarcotoxins without reference to geographic location); and Scombroid poisoning, the result of eating tuna-like fish (tuna, bonito, skipjack, mackerel) in certain tropical regions.

They appear to vary in severity, types of symptoms and species of fish. The disease can be defined as a type of intoxication resulting from the ingestion of a neurotoxin which is present in the bodies of certain fishes, and variously manifested by symptoms of extreme weakness, malaise, pruritus, myalgia, paresthesias of the mouth and extremities, paralyses, and convulsions, generally associated with such gastro-intestinal symptoms as nausea, vomiting, diarrhoea and abdominal pain. Death, when it occurs, is from respiratory paralysis.

Regarding the presence of salmonella in fresh fish abroad, Floyd and Jones (1954) isolated shigella and salmonella organisms from the intestinal contents of fresh fish sold in Cairo, Egypt. Gulasekharam, Velaudapillai, and Niles (1956), during an investigation in Ceylon examined 629 fresh fish (65 species) and 42 samples of fish washings from a market in Colombo, for the presence of salmonella: 39 fish and 5 samples of washings were found to be harbouring the organisms. Of the 15 salmonella types isolated all except 2 had been previously isolated from human and animal sources in Ceylon. The authors remark that 'the presence of Salmonella in the intestines of fresh fish examined suggested that the organisms had entered the live fish, whilst their isolation from the gills and fish washings was evidence that the fish could have been contaminated post-mortem.

Haff's disease in Sweden (Berlin, 1948): the symptoms of Haff's disease may be recapitulated briefly. About 18 hours after eating certain fish—perch, bream, roach, eel, etc.—the patient is suddenly seized with acute pain and tenderness in the muscles (except those of the head and face), the merest touch causing agony, and movement is impossible. Urine passed after the onset is brownish black in colour, with perhaps a few red and white corpuscles and granular casts. The reaction for myoglobin is positive. The symptoms pass off in 48 hours or so, leaving only slight tenderness and stiffness. There is no rise in temperature. Some patients are repeatedly attacked, one girl of 16 years is reported to have had 7 such attacks, while others partaking of the fish escape altogether. Hence it is thought that there may be some idiosyncrasy. Birds and animals eating the fish die in considerable numbers.

Investigations as to the actual cause have ruled out bacterial or virus infection, poisoning from discharge from factories, selenium, plankton, and allergy to extracts of the fish. Further study has demonstrated that certain fish contain a substance which inactivates vitamin B_1, and it is thought that Haff's disease may be the result of a disturbance in the metabolism due to the presence in the fish of an 'antivitamin' which is capable of completely suspending or reducing the biological action of aneurin.

Shell-Fish

Many persons show a definite idiosyncrasy to shell-fish generally, even when eaten in season, and urticaria and gastro-intestinal symptoms, etc., which vary considerably in individual cases, usually follow their consumption.

Apart from outbreaks of typhoid fever due to infected shell-fish collected from beds polluted by sewage, cases of food poisoning have been reported from time to time due to the consumption of mussels, oysters, cockles, crabs, lobsters, etc.

Gray (1936) records an outbreak of gastro-enteritis at Avonmouth affecting 18 persons. The illness, which was caused by the consumption of cockles purchased from an itinerant vendor, was associated with *B. Proteus vulgaris*.

Mussel Poisoning

Mussels are particularly liable to be toxic, and cases and deaths have been recorded (see Cameron, 1890; McWeeny, 1890; Todd, 1891; Hill, 1895; Kofoid, 1927, and Meyer, 1928) as being due to the ingestion of these shell-fish.

Many theories have been put forward to account for their poisonous nature. Dutertre came to the conclusion that no class of mussel was always poisonous, that the toxic action was not due to some particular food eaten by the mussel, or to spawn or any portion of the mussel itself or to decomposition, but that the poison was due to a true disease attacking the liver.

Dodgson (1928) says:

> The widely held popular view that all manifestations of poisoning, due to the consumption of mussels, arise from a common cause inherent in mussels is erroneous. Certain popular conceptions that poisonous properties reside in the 'beard', foot or other particular parts of the mussel, are erroneous.
>
> Mussel poisoning includes at least three distinct pathological conditions or types of condition, namely:
>
> (*a*) 'Musselling' or the erythematous form, which is due to properties

inherent in the mussels. It affects a limited number of specially susceptible people, who should avoid mussels. The symptoms are of short duration and unpleasant, while they last, but are never serious of import.

(*b*) The paralytic form, always grave and even fatal. Its cause is not definitely known, but it is always due to mussels from foul or stagnant waters. It is extremely rare, some 8–10 cases only being on record. The dangers of contracting it may be reduced to such small proportions as to be probably entirely negligible if elementary caution be exercised, and especially if only purified mussels be eaten.

(*c*) The bacterial food poisoning form. There are recorded in the literature several cases of fatal poisoning following the consumption of mussels, which were, in all probability, instances of bacterial food poisoning. Generally speaking, the cases in question are difficult to classify, either because the information available is inadequate for the purpose or because certain of the symptoms were such that it is not possible to exclude the paralytic form.

With regard to (*b*) paralytic form, the classical mussel poisoning outbreaks at Wilhelmshaven in 1885 (19 cases with 4 deaths) and Dublin in 1890 (7 cases with 5 deaths) well illustrate this type of poisoning. The typical symptoms were vomiting, swelling of the face, constriction in throat, numbness of mouth and lips, pricking and burning sensation in hands and feet, want of co-ordination of movements, giddiness, spasms, and dilation of the pupils of the eyes. Death resulted from respiratory paralysis.

In the Wilhelmshaven outbreak, Brieger (1889) isolated a substance he called 'Mytilotoxine' from the mussels which, when injected into animals, produced all the symptoms of mussel poisoning.

Meyer (1928), in recording an outbreak of mussel poisoning in California in 1927, where 102 persons were affected and 6 died, points out that poisonous mussels cannot be distinguished from sound molluscs, either by appearance or behaviour on cooking; occasionally a pungent odour may be noticed and the 'liver' is always large and dark. In this particular outbreak the mussels were neither located in stagnant nor polluted basins, but were subjected to the ebb and flow of the tide. Incidentally, poisoning due to the consumption of Pacific Coast mussels has been known since the days of the Indians. They noticed that if the shell-fish were eaten after being collected when the ocean waves were luminescent (in hot weather) they caused illness and death.

Somner and Meyer and their colleagues (1937), who made an experimental study of paralytic shell-fish poisoning comment as follows:

Deductions from analogy with other poisons are of little avail in the elucidation of the problem, since paralytic shell-fish poison seems to

belong to a category all its own from the toxicologic as well as possibly from the chemical point of view. In its powerful action of the respiratory centre it resembles some of the most potent alkaloids, but it far surpasses them all in toxicity.

The investigators tabulated 243 cases of paralytic shell-fish poisoning, with 16 deaths, that occurred between Ventura County, California, and Juneau, Alaska, from 1927 to 1936. Of these, 234 were caused by the coast mussel and 9 by the Washington clam. They conclude:

Poisonous mussels may in no way be distinguished from normal ones except by the animal test. Mussels subjected to various conditions in the laboratory have never shown an increase in toxicity; they usually show detoxification, the rate of which has been determined. Mussels may take up poisons from sea water. Strong evidence has been presented which points to the water of the open ocean as a carrier of the poison. Owing to the strong absorption of the substance on base-exchanging silicates of the sand, it is not likely to occur free in the water. The poison has been demonstrated, at least during the poison season, in the residue from filtration of sea-water. Whether it is contained in the plankton or absorbed in the microscopic sand cannot at present be decided.

In their summary on mussel poisoning, Somner and Meyer (1941) state:

The original source of the poison is found in a unicellular microscopic organism of the ocean, the dinoflagellate Gonyaulax catenella. It is free-swimming organism, multiplying by formation of chains of 2, 4 or even 8 individuals, of dark-orange or greenish-brown colour, and living, like a true plant cell, by photosynthesis. Like all plankton organisms it is most abundant in the summer; at times it may multiply to as large a number as 40 million per litre. At such times the water may, for miles present a deep rust-red colour, the so-called 'red-water', in daytime, and a beautiful luminescent spectacle at night. Needless to say, other dinoflagellates or diatoms may present similar pure culture developments in the ocean without being poisonous. Gonyaulax catenella may vary considerably in its poison content; even a small number which is not visible as red water may be sufficient to cause dangerous conditions in shell-fish. It occurs in the open Pacific Ocean, less in enclosed bays and estuaries, from Alaska to Southern California. It has been tentatively identified in the North Atlantic Ocean (Nova Scotia and Belgium).

The strong radiation of the sun together with the cold nutrient waters due to the upwellings along the Pacific Coast in summer time seems to be the ideal conditions for the growth of this dinoflagellate.

The Poison: The poison contained in this organism is one of the strongest known. It belongs to the class of alkaloids, such as strychnine, muscarine and aconitine. It is heat-stable in acid or neutral solution, but is gradually destroyed by boiling with alkali. It is readily soluble in water and alcohol, insoluble in ether or chloroform. About one millionth

of a gram is sufficient to kill a mouse on injection; the fatal dose by mouth for a man is probably a few milligrams. The toxic principle has not been isolated in a crystalline state but has been purified to a high degree in the form of its hydrochloride.

Medcof, Leim, Needler, Needler, Gibbard, and Naubert (1947) recorded the results of a survey made of the commercially important shell-fish areas of the Maritime Provinces on the Canadian Atlantic Coast, which demonstrated paralytic shell-fish poison in shell-fish only from the bay of Fundy area.

Samples of shell-fish were collected from typical suspected areas and examined. The undermentioned six species were found to be affected.

Common Name	*Scientific Name* *Johnson* (1934)	*Relative Toxicity*
Red or horse mussel	*Modiola modiolus* Linné 1758	10
Blue, black, or common mussel	*Mytilus edulis edulis* Linné 1758	10
Bar clam	*Mactra solidissima solidissima* Dillwyn 1817	9
Razor clam or razor fish	*Ensis directus* Conrad 1843	7
Smooth or sea scallop	*Pecten grandis* Solander 1786	1
Soft-shell clam	*Mya arenaria* Linné 1758	1

The implication of the soft-shell clam is most important from an industrial, as well as from a public health point of view.

There is one seriously dangerous period annually and it recurs in late summer and early fall. The degree of toxicity and length of danger period vary from year to year. The dinoflagellate, *Gonyaulax tamanensis*, Lebour (1925), on which the shell-fish feed (samples of the dinoflagellate were submitted and identified by Miss M. Lebour of the Marine Biological Laboratory, Plymouth, England) is the only plankton observed so far, whose periodic increases in abundance coincide with the appearance of high toxicities. Its swarmings are apparently controlled by water temperature. It is strongly suspected as being the ultimate source of the poison in the Fundy area.

The evidence presented indicates a wide range of human susceptibility to paralytic poison. Some people appear to be susceptible while others seem to have a natural tolerance to considerable amounts of the poison and others to have acquired tolerance.

Ordinary domestic cooking of poisonous shell-fish reduces the danger of poisoning but does not provide sufficient protection.

Commercial canning is much more efficient in this respect, but shucking has no demonstrable effect. There is no ready means of detoxification of raw shell-fish that can be practised commercially.

Sapieka (1948) investigated the cases of several persons taken ill after eating the white mussel (*Donax serre*) and also the black mussel, readily obtainable in South Africa. An extremely toxic alkaloid could be extracted from the liver and flesh of the mussels. The source of the poison appeared to be a type of plankton on which the shell-fish feed. It multiplies so rapidly that the water becomes a deep red colour. This has been observed during the fortnight preceding the outbreak referred to.

Gastro-intestinal infections are conveyed at times by shell-fish. It is therefore essential to protect the shell-fish in growing areas, as well as the area adjacent thereto from becoming infected with the organisms known to be conveyed by excretions from the human body.

Salmonella in Mussels

Macdonald, Sivell, Emms, and Douglas (1948) examined mussels obtained from Brancaster Staithe on the North Norfolk coast for the presence of pathogenic organisms. They isolated a new type of salmonella, subsequently referred to as *Salm. brancaster*. It was not isolated, however, from any case of human infection. The investigators state:

> Though shell-fish have been responsible for many outbreaks of typhoid fever, they appear to have caused relatively few cases of paratyphoid or other salmonella infections. In a limited experience we have recently isolated *Salm. typhi-murium* and *Salm. newport* from mussels, but in each instance the number of pathogens was very small. With the increasing use of shell-fish as human food there may be more opportunities for cases to become infected from this source.

Subsequently Hinder, Taylor, and Walther (1952) described an outbreak of gastro-enteritis due to *Salm. brancaster*, which occurred in a children's ward in a hospital.

Gemmill (1957) describes two interesting outbreaks of mussel poisoning of the gastro-intestinal type, which occurred in Glasgow City during the months July—September 1950 and 1953 respectively. In all, 80 persons were affected, but no deaths occurred. The incubation period varied from 1 to 17 hours and duration of the illness 1 to 4 days. The symptoms included nausea, vomiting, abdominal pain, diarrhoea, and in some cases headache, fainting, and prostration. Samples of faeces and urine were collected from 40 patients and submitted to bacteriological examination, but no pathological organisms were isolated.

The mussels were collected from sites on the inland sea-boards of two lochs, the Gareloch and Loch Long, some 30–40 miles from Glasgow. The Gareloch receives the crude sewage, not only from surrounding villages but also from Helensburgh, a town of about 9,000 population, which is greatly augmented by visitors in summer. Warning notices regarding shell-fish are displayed along the foreshore. Loch Long is in a more remote and less populous area. One of the sites situated at the mouth of a stream contained a sewer outfall, which was in an especially unfavourable position. The supply of mussels from the lochs was stopped and the outbreaks ceased.

Gemmill remarks:

Both outbreaks were of a mild gastro-enteritic type. Judged by the incidence of prostration, the illness was rather more severe in 1950, but the pattern in both was very similar. From the facts elicited by investigation, it may reasonably be concluded that the outbreaks were caused by a heat-stable toxin associated with bacterial contamination, that the mussels were already contaminated before they left the sea-bed and that their poisonous state could not be attributed in any way to the restaurants nor to the presence of a metallic agent.

The Purification of Mussels and Oysters

Sherwood (1947) says that any effective remedy for pollution must meet these requirements:

1. For the consumer of shell-fish it must eliminate risk of any illness or poisoning (other than 'musselling' of those susceptible to it), and in so doing safeguard the public health to the satisfaction of the authorities concerned under the Shell-fish Regulations.
2. There must be means for identifying shell-fish which have been rendered safe, and when tested bacteriologically such shell-fish must show a satisfactory degree of freedom from faecal contamination.
3. The flavour, condition and keeping quality of the shell-fish must remain unimpaired.
4. The remedy must be simple and certain in action at all times on a readily standardised routine basis.
5. It must be economically sound. This means in practice that efficiency must not depend on close scientific and bacteriological control, nor must the cost be unrelated to the value of the shell-fish.

A comprehensive series of experiments were carried out at the Fisheries Experimental Station (Ministry of Agriculture and Fisheries), Conway, North Wales, under the direction of Dr. Dodgson (1915), in order to establish a reliable treatment for the purification of mussels.

The system of purification was based on the comparatively simple and natural action of the bi-valves clearing their alimentary

canals freely in uncontaminated (chlorinated) sea-water, after a preliminary hosing down to clean the shells. The purification process was found to be satisfactory. Bacteriological examination of the mussels after treatment revealed that not only did they rid themselves of sewage and bacteria, but also of all particles of solid matter during the 48-hour treatment.

After the installation at Conway had been approved by the Ministry of Health, it was taken over by the Fisheries Department (1918). Later, similar installations for the treatment of mussels were put into operation at Lytham, Lancs., and also at Killorglin, Cromane, in south-west Ireland.

With regard to oysters, experiments were carried out at the Fisheries Experiment Station, Conway, and it was found that these molluscs could be purified on a commercial scale in a similar manner to mussels; but that a special device was necessary in order to cleanse the outsides of the shells. A temperature of 56°F. had to be maintained, however, during the process. In 1934, oyster purification on a commercial scale was carried out at Brightlingsea, Essex.

The mussels and oysters after treatment are packed in sterilized bags or containers, each being secured by a lead seal indicating origin and date of dispatch.

It should be specially noted that the shell-fish thus purified are not impaired either in keeping quality or any other respect. Thus the problem of safeguarding the public health from infection from contaminated mussels and oysters is practically solved.

Experiments were carried out at Lytham, Lancs., Mussel Cleansing Station, in 1948, to ascertain the possibility of repeatedly using the same sea-water for the treatment of shell-fish from polluted areas, to cleanse them of bacteria. The sea-water was treated with sodium hypochlorite and then aerated to re-oxygenate the water. It was concluded that the sea-water after such treatment could be re-used for the whole season (Water Pollution Research Board, 1949).

Allen, G. Thomas, M. C. C. Thomas, Wheatland, H. N. Thomas, Jones, Hudson, and Sherwood (1950) in their investigations point out that:

when mussels are allowed to function in sea-water the main changes occurring in the water are depletion of dissolved oxygen and lowering of *p*H value. Provided the faeces and pseudo-pieces are not disturbed, the increase in the content of organic matter is not appreciable. If the supernatent water is removed and aerated with diffused air the water is

re-oxygenated, the *p*H value is restored to its original level and the water so treated may be re-used for immersing a fresh batch of mussels. The process of re-use may apparently be continued indefinitely.

They advise, however, that the chlorination of the sea-water take place between each cleansing of the mussels and that the temperature of the re-used water be maintained at 6°C. (43°F.) or above. Oysters could be cleansed in the same way, but the temperature of the water should be maintained at 12·2°C. (54°F.) or rather higher.

Recent researches into the purification of oysters (Cole, 1954), have shown that these molluscs (English and Portuguese), purify themselves when placed in simple constructed shallow pits containing not more than 2 feet of sea-water without previous chlorination, or any other form of sterilization, for a period of 4 days at any temperature between −1° and 25·2°C. The oysters before being placed in the pits are cleansed of mud, barnacles, etc., adhering to the shells.

The results of the bacteriological examination of the treated molluscs proved that *B. coli* were absent in 1 ml. of oyster flesh after 4 days in the pit, showing that a satisfactory state of purity was obtained by this simple method. The oysters before being packed for the market are cleansed by placing them in a wire basket and rinsing in a tub or pit of water, or by hosing with water which is allowed to run to waste. If a special pit is maintained for washing the oysters, the water must be either chlorinated or allowed to stand for 4 days before it is used, to allow any faecal coli to die out (Vaccaro, 1950).

Wood (1957) reports that the results of experiments carried out at Brightlingsea, Essex, to ascertain the temperature and time necessary for satisfactorily cleansing oysters, 'show that cleansing is complete within 48 hours and that the temperature of 5°C. (41°F.) later selected for full scale process, would offer a sufficient margin of safety'. This cold-water cleansing process was in operation during the winter of 1955–6 and weekly sampling showed the oysters to be highly satisfactory. Moreover, a considerable saving in overhead costs resulted as the water was only warmed for a few weeks instead of regularly each year from September to May.

Ozonization treatment for the purification of oysters is also in use at a number of sea fisheries. Briefly, the process consists of passing sand-filtered, ozonized sea-water continuously over trays (fitted in wooden vessels) containing the oysters, which are cleansed in from 48 to 72 hours. The treatment is effectual, cheap, and economical.

Bacteriological Standards

There is no legal standard for the bacteriological cleanliness of shell-fish in this country. Of the many standards used, that of the Fishmongers' Company allows about 100 coliform organisms per oyster on 40 per cent of the specimens examined. A more satisfactory bacteriological standard has been suggested by Clegg and Sherwood (1948) who measure the degree of contamination of faecal coli in a single test which does not require confirmation. Roll-tubes of a modified MacConkey agar are inoculated with material from the shell-fish and incubated at 44°C., a temperature which inhibits the growth of non-faecal coli. If 4 out of 5 samples of shell-fish from the same source are free from faecal coli in 1 ml. quantities of tissue such shell-fish are said to be fit for food. If there are 2 or 3 faecal coli per ml. of shell-fish in any sample further investigation is required.

In the Fishmongers' Company Memorandum, Knott (1951), three methods for the examination of oysters and other shell-fish are reviewed in detail, namely: the short method (Klein and Eyre), the long method (Houston-Eyre), and the combined roll-tube and percentage method (based on that of Clegg and Sherwood). On the basis of experiments then completed it was suggested that a bacterial coli count standard and the company's 'Percentage Clean' standard should correspond approximately as follows:

Percentage Clean	*Bact. coli (Faecal Type I) per ml. of flesh*	*Conclusion*
100 to 80 per cent	5 colonies	Quite satisfactory
70 per cent	5–15 colonies	Suspicious. Further samples to be taken. Sale not immediately prohibited.
60 per cent and lower	Above 15 colonies	Unsatisfactory. Sale prohibited until further samples examined.

Sherwood and Scott Thomson (1953) in their summary on the bacteriological examination of shell-fish as a basis for sanitary control, point out:

Shell-fish which have passed through the approved tank process or have been gathered from unpolluted sources cannot be guaranteed free from faecal coli, and a standard demanding this would be unreal and artificial. Samples containing not more than 5 faecal coli per ml. are as

clean bacteriologically as uncooked shell-fish can be expected to be, and for many years the special treatment centres have regarded this result as confirmation of satisfactory treatment.

They suggest three grades of purity of shell-fish, as follows: Grade I—Not more than 5 faecal coli per ml. of shell-fish. Grade II —6 to 15 faecal coli per ml. Grade III—over 15 faecal coli per ml. 'Samples now examined in laboratories are not representative of the shell-fish offered for sale in all parts of the country. Whether the suggested standards are reasonable will not be known until samples are more widely taken for examination.'

Sherwood (1957), carried out experiments on the sterilization of cockles and mussels by boiling. In his summary he states:

> In experiments on heating shell-fish in a relatively great volume of water, to avoid cooling on immersion, sterilization of large cockles and mussels resulted from immersion for two and a half minutes respectively at 100°C. For each reduction of water temperature by 10°C. down to 70°C., extension of the immersion period by about half a minute was necessary. It is suggested that to make polluted cockles and mussels safe for human consumption by heating in bulk they should be kept fully immersed in boiling water for one and two minutes respectively after resumption of boiling.

Characteristics of Molluscs and Crustaceans

The shell of a living bivalve is usually tightly closed. If found open, it should close immediately when handled, otherwise it is dead and unfit for food. Oysters should be eaten as soon as possible after being opened. The close season for natives is from 14 May to 4 August, and for deep-sea oysters from 15 June to 4 August. The season for mussels is between 1 May and 30 August.

Scallops: The shells may sometimes widely gape though the animal is alive, but if the surrounding mantle (curtain) is touched, a distinct movement is noticed, denoting that the scallop is alive. Staleness and decomposition are detected by smell. The flesh should be white and firm and roe orange in colour. These shell-fish are in prime condition from December to March.

Cockles: When fresh and sound the shells of cockles are tightly closed and difficult to open. If found open, a tap on the shells should cause them to close. Gaping shells and offensive smell are sure signs of unsoundness. The shells may remain closed even when the shell-fish is dead. When opened, however, the fish will be found thin, dry, or decomposed. Cockles are in best condition from June to December.

The most important industries are carried on at Leigh-on-Sea

and Southend-on-Sea. There is a large demand for these shell-fish during the summer months. Regarding purification, the method of cooking cockles comes under the supervision of the Port Health Authority, under the Public Health (Shell-fish) Regulations, 1934. Control is exercisable by the Local Authority under Sec. 13 of the Food and Drugs Act 1955. Cockles have been from time to time the cause of outbreaks of illness. One outbreak occurred in 1946 due to *Salm. typhimurium* and another in 1949 (398 cases), recorded by Stevenson, Logan, and Oliver (1952).

Pilsworth (1952) in his bacteriological studies on cooked shell-fish, remarks:

> The Southend outbreak of 1949 ascribed to cockles would appear to be characteristic of the type caused by 'non-specific' bacterial agents and associated with high temperature and poor hygiene, rather than the type due to a specific organism associated with the presence of human excreta.

Periwinkles: The foot of a fresh winkle is prominent and not shrunken. The horny door (operculum) is moist and clean and the flesh does not break when removed from the shell. Smell is the best guide as to freshness. In commencing decomposition they feel clammy and odour is offensive. Periwinkles are in season throughout the year and are usually boiled for about 8 to 10 minutes before sale.

Crustaceans—Crabs and Lobsters: Boiled crabs do not keep in good condition many days, especially during warm weather. When fresh, the joints of the abdomen and limbs are intact. If stale, the shell is faded and dull looking and later becomes soft. The limbs are easily detached and a sticky substance oozes through the inner covering at the joints. There is discoloration of the apron and an offensive smell. Crabs are in season all the year round but are in the best condition during April, May, and June.

Harris (1932) proposed the Nessler ammonia test as a means of differentiating fresh from spoiled crab meat, as follows. About 1 gm. of crab meat is shaken up with a small quantity of distilled water. To this is added 2 or 3 drops of Nessler's reagent. If a deep yellow or brown colour develops, spoilage of the meat is indicated.

The indication of a good and fresh condition in the lobster is a bright, hard, clear shell, with the flesh plump and firm. After being cooked, the tail on being pulled out should spring back sharply, but when stale it loses its springiness. Lobsters are in first-class condition during the summer months. Signs of decomposition are the same as those for the crab.

Shrimps and Prawns: Shrimps after being boiled have a fresh pleasant smell. They should be dry and crisp and have a clean-looking shell. The stiff tail is pressed inwards towards the body. When stale, and as decomposition advances, heat is evolved, the shrimps become soft, sticky and slimy, and the smell is musty and unpleasant. The above characteristics also apply to prawns.

Acts and Regulations Relating to Fish and Shell-Fish

Sea Fisheries Act, 1868 (amended by Sea Fisheries Act, 1884).
Fisheries (Oyster, Crab and Lobster) Act, 1877, amended by The Sea Fishing Industry (Crabs and Lobsters) Act, 1951.
Sea Fishing Industry Act, 1933, amended by the Sea Fishing Industry Act, 1938, and further by the Sea Fish Industry Act, 1951.
Public Health (Shell-Fish) Regulations, 1934 and 1948.
Sea Fish Industry Act, 1938, amended by the Sea Fish Industry Act, 1951.
Sea Fishing Industry (Immature Sea-Fish) Order, 1948.
Sea Fish Industry Act, 1951.
Food and Drugs Act, 1955 (Sec. 25).
Food Hygiene Regulations, 1955 (Part IV, 25).

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Chapter XV

FOOD ALLERGY

The existence of peculiar abnormal reactions or idiosyncrasies or sensitization to food has been recognized since ancient times. Many persons are unable to eat certain foodstuffs, even in very small quantities, without exhibiting some characteristic and/or disagreeable symptoms; moreover, the number of food-sensitive persons is, in all probability, much greater than commonly suspected. It was not, however, until the beginning of the present century that any real understanding was reached about the mechanism which produced the condition. The old quotation, *Quod aliis cibus est aliis fuatacer venemum* (Lucretius, 96–55 B.C.) —literally translated 'What is food for some may be strong poison for others'—probably originated from the knowledge of these idiosyncrasies. Physiologically this peculiar and interesting sensitivity to particular foods, termed 'food allergy' (Clemens von Pirquet, 1906), which is fairly common and sometimes of a more or less serious nature, is primarily due to the constitutional condition of the individual concerned and not to the result of eating unwholesome foodstuffs. 'Allergy is a general term applied to any alteration in the reaction of the living organism to foreign substances; it may be antigenic or non-antigenic in character' (Jordan, 1931).

Charles Richet coined the word 'anaphylaxis'. Vaughan suggested that anaphylaxis and immunity depended on the same fundamental process. This theory is usually accepted as anaphylaxis and allergy are basically very similar. They are the result of the same kind of physiological reaction, except that the former is more acute.

Much light was thrown on the allergic process by Dale and Laidlaw in 1911. They demonstrated that in the course of an allergic reaction histamine and histamine-like substances were released and that many of the allergic manifestations could be reproduced by histamine.

The foods which commonly produce these allergic reactions usually contain proteins or are protein in nature (nitrogenous foods), and include a large number and variety of articles of diet, such as eggs, milk, cocoa, cheese, fish, shell-fish, cereals, peas, beans,

nuts, onions, cucumber, radishes, potatoes, pork, strawberries, blackberries, mushrooms, etc., or a combination of several of these commodities. Moreover, there is some evidence that non-nitrogenous substances (oils, fats, and carbohydrates) also may be responsible. The diagnosis is established by elimination and trial diet methods.

With regard to fish, even the odour of cooking is sometimes sufficient to induce symptoms in highly sensitive persons. Cooking has little effect upon fish allergy. Cereals, which are common ingredients of the average diet, have many commercial uses. They are of special importance because of their ability to cause symptoms, either as a result of inhalation (as asthma in bakers) or when ingested, being capable of producing almost any variety of allergic manifestations.

Rarely is a person sensitive to only a single food, but generally sensitive to a group of similar foods. Investigators who studied the subject extensively found that next in frequency come chocolate, cabbage, tomatoes, oranges, cauliflower, bananas, walnuts, carrots, raspberries, blackberries, and peaches. The manifestations in connection with food allergy vary considerably in individual cases. Sensibility to the reactions can be influenced by some special condition of the person concerned (such as fatigue, nervous excitement, worry or other emotional disturbance) at the time when the particular food or combination of foods is consumed. The onset of the attack may or may not be sudden and its duration varies from a few hours to a week or more. The reactions, which can be mild or severe, include: nausea, vomiting, gastro-intestinal disturbance, constipation, migraine, urticaria, erythema, eczema, rhinitis, pruritus ani, bladder allergy, and certain forms of malnutrition. Very mild reactions, which are the commonest, sometimes produce symptoms so slight that their true nature may be overlooked. These mild reactions may also give rise to recurring illnesses, or chronic ill-health. Adult women are slightly more prone to allergic manifestations than men.

Foods to which an individual may be sensitive do not always produce the same manifestations. For instance, one may cause an urticarial rash and another a gastro-intestinal disturbance. Occasionally the mere handling of a certain foodstuff, such as flour (or even drugs), by very sensitive persons, is sufficient to set up localized reactions, particularly skin affections, such as eczema. Special exposure enormously increases the incidence of sensitiveness.

Several of the above named reactions are often present in various types of food poisoning, especially isolated cases with gastro-intestinal symptoms, which are more likely to be due to food allergy, than are large outbreaks, thus adding to the complexity of the whole subject.

Age has an important bearing on the incidence and possibility of food allergy. It is commonly present in infancy, tends to grow less as age advances, but may be acquired at any time during life as a result of excessive consumption of some particular or unusual food such as, strawberries, mushrooms, etc. Ratner (1928) suggested that under certain conditions an infant with an allergic predisposition may be sensitized before birth by the mother's over-indulgence in certain protein foods. Rubin (1940) records four instances of allergic melena in newborn infants. The condition may or may not be hereditary, and there appears to be a considerable difference of opinion on this subject though hypersensitiveness exhibited towards certain foods is frequently present in parent and offspring. An inherited tendency to become sensitive to certain foodstuffs may show itself at any time after birth, but it does not necessarily follow that descendants will suffer from the same allergic manifestations as their antecedents. The tendency to become sensitive, however, is no doubt transmitted from one generation to another. A case has been recorded (Richet, 1913) where idiosyncrasy to eggs existed in four generations.

In infancy great difficulty is frequently experienced in feeding, and this is increased by the presence of sensitization to common foods, such as milk, eggs, and even human milk. Sensitivity to eggs, more than other foods likely to cause allergy, is provocative of infantile cutaneous manifestations such as eczema and urticaria. Children showing an idiosyncrasy to cow's milk can often drink goat's milk with impunity. A definite early history of dislike for, or avoidance of, some particular food or of disturbances caused by them, may sometimes suggest the presence of this hypersensitive condition.

It must be remembered that milk is commonly used in the preparation of a large number of foods such as custards, cakes, ice cream, macaroni, spaghetti, cream soups, sauces, infant foods, milk chocolate, and many other articles which may not be suspected by those persons who are subject to allergic reactions from the consumption of even the smallest quantity of milk. The majority of hypersensitive persons are usually only allergic to raw milk, but there are others who are affected by pasteurized or heated milk.

Rowe (1931), in a series of 175 cases, found 57 per cent due to wheat, 31 per cent due to milk, and 35 per cent to eggs. Ratner (1935) in his studies in milk allergy showed that heat elements in milk (lactalbumin and lactoglobulin) are responsible for practically all the cases, so that some form of heat treatment can be safely used. Alvarez (1937) in a group of 500 cases due to food allergy, found 26 per cent were due to milk, cream, and ice-cream.

Regarding the general issue of milk to schools, Kennedy (1936) remarks that 'the widespread drinking of milk by school children and others may yet have to be considered in the light of food allergy.' Williams (1936) studied children who refused or showed no inclination to drink school milk: 58 children refused because its ingestion was followed by allergic manifestation, generally gastro-intestinal.

A characteristic feature of food idiosyncrasy, especially in young children, is the tendency for one reaction to be replaced by another. It is quite possible for an infant with severe urticarial rash to outgrow this manifestation and in later years to become subject to gastro-intestinal symptoms. In certain individuals the periods of sensitivity may be separated by periods of lessened sensitivity when they are comparatively free from attack. In some persons the abnormal condition may gradually become continuous, while in others the reactions may actually disappear altogether in course of time.

Dodgson (1928) described an attack of 'musselling' he had when a young man. About 24 years later he again ate mussels with the intention of recording the symptoms. Nothing however happened.

Although the real cause of food allergy is probably not fully understood, the abnormal condition has been generally assigned to individual hypersensitiveness to foreign protein substances circulating in the blood. It is well known, as a result of experiments, that the injection of protein substances into man and animals may at times set up poisonous symptoms. Some observers hold the view that this hypersensitive state may be due in the first place to an abnormal permeability of the intestinal mucous membrane, which allows the unaltered proteins to pass through in an unchanged state and in this way gain access to the bloodstream. The condition resembles in certain respects the condition known as anaphylactic intoxication, which is presumed to be an exaggeration of the normal defence of the body against proteins and bacteria through the agency of the processes of digestion.

Savage (1920) remarks:

> The hypothesis that these cases of food idiosyncrasy are a variety of anaphylaxis is based on the supposition that in the individuals who exhibit the condition there is a marked hypersensitiveness to the action of particular proteins in these special foods, that they gain access to the circulation as unaltered protein and that the symptoms caused are due to individual intolerance of their presence in the blood. There are strong arguments which suggest this is the true explanation. In the first place the symptoms induced, including the rapidity of onset (allowing time for absorption from the alimentary canal), the minute dose required and the lesions caused, resembled in many ways those recognised as symptoms of anaphylaxis.

Here are interesting and illustrative cases of hypersensitivity to certain common foods. One recorded by Talbot (1916) deals with milk. Reactions due to proteins in this food are fairly common. A healthy baby, which was breast-fed till it was 8½ months old, was given cow's milk and barley water without any ill-effects. This was stopped for a few weeks, but when cow's undiluted milk was added to the diet the child vomited and showed decided symptoms of illness and within an hour its body was covered with an urticarial rash. Substitution of goat's milk for the cow's milk at once stopped the trouble.

Hazen (1928) records the case of a young woman (19 years of age) who had suffered from chronic dermatitis since the first year of her life, except when she was living on an island where milk was unobtainable. Minute traces of cream caused the skin manifestations. She remained free from the dermatitis as long as milk was entirely avoided.

Cases of hypersensitiveness to egg albumen frequently occur and several typical instances have been recorded. Coues (1912) described a case where a child about one year old was given the white of an egg which immediately caused nausea and vomiting. About 8 months later the child was again given white of egg. Violent sneezing and all the symptoms of an acute cold in the head followed, an extensive urticarial rash appeared on the body, and the eyelids became oedematous. The temperature remained normal and there was no marked prostration.

Jordan (1931) in reference to sensitization to egg albumen states that in some cases the amount of the specific protein that suffices to produce the reaction is exceedingly small. One physician writes of a patient who 'was unable to take the smallest amount of egg in any form. If a spoon was used to beat eggs and then to stir his coffee, he became very much nauseated and vomited violently.'

Hypersensitiveness to white of egg is of particular significance because mere traces of it are capable of causing manifestations of great severity.

Kennedy (1936) recorded a peculiar case of allergy in a woman, caused through the consumption of chocolate. She had recurring eczema on various parts of the body but was otherwise healthy. When put on a special diet the eczema completely disappeared within a fortnight. Later, however, the disease recurred and strict inquiry revealed that she had eaten chocolate. She heeded a warning not to do so again and her skin got quite well and she was able to take all foods. A piece of chocolate was then given as a test with the result that the eczema reappeared.

Lefevre (1930) described a case of illness in a soldier which may have been due to sensitization. After eating pineapple he showed symptoms of vomiting, pain in the stomach, and finally lost consciousness. Two others who partook of some of the same pineapple were not affected. This type of food idiosyncrasy has been described on several occasions. McBride and Schorer (1916) collected particulars of 60 cases of food sensitiveness causing skin trouble, such as urticaria and erythema. Fish, tomatoes, cheese, and eggs were among the foods causing urticaria, and cereals and pork the erythema, the illness appearing within less than 4 hours after consumption of the food. Tomatoes and cereals generally produced these conditions in less than 1 hour, the eruption lasting from 1 to 12 hours and in a small percentage of cases from 1 day to 1 week.

In a study by Randolph and Yeager (1947) of 200 consecutive cases of food allergy in the United States it was found that there were 87 cases of corn sensitivity, 85 of wheat, 75 of milk, and 67 of eggs. This confirmed the earlier work of Rinkel (1940).

With regard to the incidence of food allergy, the only definite figures available are the result of a questionnaire issued to 400 students and nurses as to the presence of allergy in their personal and family histories. The result suggested probable food allergy in more than 30 per cent of all persons.

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PART IV

Chapter XVI

BOTULISM

Historical

THIS comparatively rare but extremely interesting type of food poisoning (German *Botulismus*, from Latin *botulus*, sausage; sometimes known as Allantiasis, Ichthyosis, or *Würstvergiftung*) apparently had its origin on the Continent. According to historical records, 'sausage poisoning', as it was then termed, was prevalent in Germany as long ago as 1735, and was at first believed to be due to contamination of the sausages by the copper and lead vessels in which they were prepared (Müller, 1735–93) and Kerner (1755–89).

The earliest recorded outbreak which attracted the attention of the medical profession occurred in Wildbad, near Würtemburg, in 1793 (Müller), and affected 13 persons, 6 of whom died as a result of eating *Schweinsmagen* or *Blünzen* (blood puddings or visceral sausages).

The characteristics of the fatal nature of the illness were brought to the notice of the court physician, Keiser, who suspected belladonna poisoning, owing to the symptoms somewhat resembling those caused by this vegetable poison. At Hofe Mosburg, in 1799, an outbreak occurred involving 5 persons, 2 of whom succumbed. The son of the family was accused of mixing henbane seeds with the sausages with criminal intent.

Subsequent outbreaks caused the authorities to realize how unsatisfactory were the methods of preparing cheap meat foods. In 1802 Jaeger published an official warning from Stuttgart pointing out the dangers arising from the consumption of unwholesome sausages and other 'made-up' meat foodstuffs and issued instructions for their proper manufacture. He suggested that the toxic action of the sausages must have been due to the presence of some vegetable seeds or spices and not to any mineral poison. In spite of the warning, however, sausage-poisoning increased in Würtemburg, and cases, some fatal, occurred in parts of southern Germany. Ostertag (1907) comments upon this distribution:

If we ask why botulism occurs so frequently and causes so many deaths in Würtemburg, an explanation is to be found, in the first place,

in the great development of sausage manufacture, and in the consumption of sausages in Würtemburg, and also, in the ignorance previously exhibited in preparing certain kinds of sausage as 'leberwürste' and 'blutwürste,' for consumption at a considerably later date. I emphasise the word 'previously' for the gradually diminishing number of cases of sausage poisoning in the last decades proves that a change has taken place in this regard. In Northern Germany, on the other side of the Main, it is the custom to eat sausages prepared from the viscera, as, for example, 'leberwürste' and 'lungenwürste,' only in a fresh condition. At any rate, smoked 'leberwürste' in Northern Germany is exceedingly rare, except in Thüringen. The so-called long-keeping sausages of Northern Germany, which are the only kinds which are preserved for the period of months or one year, consists of musculature, which, when properly conserved, resists decomposition much longer than lungs, liver or blood. In the etiology of sausage poisoning in Würtemburg, however, smoked visceral sausages play an important rôle. These sausages are poorly adapted for keeping for a long time, since they contain material which spoils readily.

During the years 1820–2, Justinus Kerner, a noted physician, made important and systematic investigations into the cause of sausage poisoning and carried out numerous experiments to prove his theories. Later, he published two monographs in which he related the history of the malady and referred to epidemics in other parts of Germany. Subsequently, laws were enacted and the disease was made notifiable. Records reveal that during the period 1735–1874, 920 cases with 366 deaths occurred in Würtemburg (Meyer, 1928). In northern Germany the malady was comparatively rare. Nevertheless, in 1822 and 1828 the Royal Imperial Government at Arnsberg issued public warnings against the consumption of semi-solid, sour, and malodorous sausage.

Leighton (1923) remarks: 'Altogether there would appear to have been about 1,200 cases of botulism in Germany during the past 130 years, with a mortality of 360, or 30 per cent.

From time to time cases of sausage poisoning continued to be reported from Anhalt, Baden, Bavaria, Hessen, Holstein, Prussia, Pomerania, Posen, Saxony and in the Provinces of Hanover and Silesia. Records also show that the malady was present at times in Austria, Denmark, Holland, Hungary, Russia, and England.

With regard to the disease in England, it may be of interest to note in passing that John Tribe (1860), Medical Officer of Health for Hackney, London, mentioned cases of sausage poisoning. He remarks: 'Medical literature in this country contains but few records of cases caused by diseased or putrefying meat.' He quotes from Taylor's work on medical jurisprudence. Taylor, when discussing poisoning by cheese and sausages, observed: 'Although

these articles of food have frequently given rise to symptoms of poisoning in Germany, there is, I believe, no instance of their having proved fatal in England.' Later in his work, however, Tribe gives an account of three fatal cases of poisoning from eating pig's liver sausages.

The majority of the earlier outbreaks of sausage poisoning in Central Europe was caused by the consumption of certain prepared meat foods, such as blood sausages, *Blutwürste* (Jaeger, 1802); liver sausage, *Leberwürste* (Von Autenrieth, 1815); *Schlackwürst*, made from pork, veal and calf blood, and *Presskopf* prepared from livers or tongues and hog's heads (Horn, 1830). These popular foods among the poorer classes were often eaten raw or partly cooked and sometimes in a semi-decomposed condition. Scientific observers naturally supposed that such foods were the primary cause of the disease, and for many years their investigations and experiments were based on this supposition. As time went on, however, observations proved that similar symptoms and illness were produced by the ingestion of many kinds of food other than spoiled sausages. They included smoked pork (Hauff, 1829), cheese, fats, and ham. The latter foodstuff was responsible for a large number of cases of poisoning recorded by observers over a considerable period.

Von Autenrieth (1833–5) drew attention to the similarity of fish poisoning in Russia to sausage poisoning in Würtemburg. Many such outbreaks of *fisckyergiftung* in Russia (Jaechnichen, 1850), Schlossberger (1852) and elsewhere (Bohm, 1876) were reported from time to time and caused much discussion. Proof was forthcoming through Madsen (1912) who, in investigating an outbreak of fish poisoning in Oro, Denmark, isolated a bacillus identical with *B. botulinus*. The patients exhibited the characteristic symptoms of botulism after the ingestion of a pickled mackerel. One of the cases terminated fatally. The fish had a rancid odour, and the toxin in the brine was neutralized by the antitoxin prepared against the bacillus isolated by Van Ermengem at Eczelles.

Innumerable theories were advanced from time to time to account for the poisonous nature of the incriminated foods; these included chemical poisons, putrefactive alkaloids, ptomaines, ferments, vegetable organisms, moulds, and other low forms of life.

As a result of constant experimental work by Kerner, it was established that the poison was developed within the sausage and was not caused by outside agents. It is a significant fact that

Kerner (1824) came to the conclusion that the exclusion of air from the sausage was necessary for the production of the poison, and that sausages in large casings (incompletely filled) made smoking difficult and were more poisonous than meat sausages enclosed in small casings. This was confirmed by other investigators. Kerner concluded that the odour was not that of ordinary putrefaction. The taste was described as sour, bitter, and burning. He suggested that cooking the sausage might inhibit the action of the poison.

Schlossberger (1852) noted that meat sausages, which were expensive and were consumed by the wealthier classes, were usually packed in small casings and prepared under cleaner and better conditions and rarely poisonous. He also observed that the poisonous sausage had a peculiar cheese-like odour.

Later, as a result of experiments, Cormack and Corneliani (1852) demonstrated that during the process of smoking the sausages, a poisonous acid (pyroligneous) was given off when burning certain woods. This acid, they believed, accumulated in the sausages and caused poisoning.

Emmert and Kuhn (1824) suggested that the fatal cases might be caused by the formation of prussic acid, owing to the blue-black colour of the blood found in the bodies of persons who had died from sausage poisoning. This theory, which was contested by other workers, caused Kerner to carry out further experiments with the result that he found the symptoms produced by prussic acid were quite unlike those caused by sausage poisoning. Moreover, prussic acid could not be detected in the blood or tissues from the fatal cases.

In the course of his many researches in the hope of finding the toxin substance, Kerner isolated a fatty acid substance from decomposed sausages and rancid fat which he termed *Leichensaüre* and believed this to be the true toxic agent, as it produced symptoms of sausage poisoning in animals, similar to those seen in the human cases.

Buchner (1823), Kastner (1823) and Horne (1828), Dann (1828) and other workers, carried out a series of investigations and experiments with fats and acids but came to no satisfactory conclusions, and Kerner's theory was not confirmed.

Bodenmüller (1834) and Krugelstein (1839), after studying the subject, were of the opinion that sausage poisoning was not due to winter-prepared unsmoked *Leberwürste*, but that the poison was produced by the methods commonly employed in Würtemburg in smoking and heating the sausages. Liebig (1843) believed that the

disease was due to the poisonous action of a ferment. Schlossberger (1852), however, was not in agreement with this, and suggested that the disease was caused by the presence of certain organic bases of the alkaloidal group.

Heller (1853) was the first to suggest that a microscopic vegetable mould inside the sausage was responsible for the formation of the poison, while other investigators (Van den Corput, 1855, Wittig, 1856, and Kasper, 1858) opined that moulds, algae (Sarcina botulina), were the probable cause of the illness, but no conclusive evidence was forthcoming. In 1886 Von Aurep advanced a theory that sausage poisoning was caused by a putrefactive base (ptomatoatropin) which he obtained from tainted fish. In the same year Ehrenberg isolated several putrefactive amines from a poisonous sausage, two of which he believed were responsible for the poisoning. He failed, however, to produce the characteristic symptoms in experimental animals.

Nauwerck (1886) gave it as his opinion that the substances described by Ehrenberg were of bacterial origin and isolated three bacilli from the identical sausage, one of which liquefied gelatine and caused putrefaction of sterile blood. Later, Redner isolated a similar bacillus from the intestines of a hog. Nauwerck concluded that this organism was ingested with the food and caused putrefaction of the intestines, resulting in auto-intoxication which produced sausage-poisoning symptoms.

It was not until December, 1895, that the responsible organism was isolated, and described by the Belgian scientist Émile Pierre Marie Van Ermengem of Ghent, during the investigation of an outbreak which occurred among the members of a musical society who were engaged to perform a dirge at a funeral in the village of Ellezelles (Hainault). After the ceremony the members partook of a cold repast in which a pickled ham played a prominent part. Thirty-four of the members were taken ill and 3 died. The incriminated ham, which had been pickled in brine for 4 months, had a musty rancid odour and bitter taste and was pale and partially discoloured but not decomposed. It came originally from a pig slaughtered in the previous August, and in the fresh state a part had been consumed without ill-effects. Van Ermengem isolated a large anaerobic bacillus from the remains of the ham and from the spleen and contents of the large intestine from one of the fatal cases. By cultivating the bacillus under anaerobic conditions he ascertained that a powerful and deadly toxin was manufactured in the surrounding medium. He made watery extracts from the

remains of the ham and injected it into experimental animals and produced in them the characteristic neuro-paralytic signs observed in the fatal human cases. The most typical symptoms were seen in cats.

This remarkable discovery thus proved the causal relationship of the bacillus and its toxin to the disease, and at the same time put an end to all speculations as to the nature of the poison causing the symptoms seen in the previous cases.

Van Ermengem compiled a complete list of the characteristic symptoms of the disease culled from close observation of the patients. He named the organism *Bacillus botulinus*, and the disease became known as botulism. Van Ermengem, as a result of his investigations, which were confirmed by other observers, came to the conclusion that botulism was not an infection but an intoxication, the bacillus not growing in the human body.

Kempner (1897), by means of Van Ermengem's cultures, showed that the botulinus toxin causes the development of a powerful specific anti-toxin in the body of goats.

Van Ermengem (1906) isolated another strain of *B. botulinus* during an outbreak among 12 persons at Isegham in West Flanders.

In 1900 Römer investigated an outbreak in the district of Alsfeld, Germany, due to the consumption of a pickled ham, which caused the illness of 4 persons. He isolated a bacillus similar to the one discovered by Van Ermengem.

Van Ermengem's finding was confirmed by Landmann (1904), Ornstein (1913), Schumacher (1913), and other observers. For some years after the discovery of *B. botulinus*, the disease of botulism was looked upon as rare and of academic rather than of practical importance. It was presumed that a meat product was essential for the satisfactory growth of the bacillus. In 1904, however, an outbreak of illness, described by Fischer (1906), occurred at the Alice Cooking School in Darmstadt among 21 persons, 11 of whom died within 4 to 5 days after the consumption of a cold home-canned white bean salad. The contents of the can had a rancid odour, but the beans showed little disintegration. A bacillus identical with *B. botulinus* was isolated, which yielded a toxin fatal to guinea-pigs in doses of 0·0003 c.c.

This was a typical example of an outbreak of botulism, not of meat origin, and doubtless attracted the attention of observers to the possibility of canned vegetables being suitable media for the growth of the bacillus and the deposition of the toxin.

Modern history records that botulism has been prevalent in the United States. Dickson (1917) considered that the disease was more common than is shown by the records.

In 1918, however, he remarked:

A review of the American literature reveals that very few cases of botulism have been recognised in this country, but in a survey of the available cases of food poisoning during the past 25 years it was found that there have been a number of cases in which the symptoms are more or less indicative of this condition.

Dickson (1918) quotes outbreaks recorded by Jellinek (1902), Sheppard (1907), Peck (1910), Stiles (1913), Wilbur and Ophüls (1914), Frost (1915), Lancaster (1916), Curfman (1917).

In America botulism has been mostly associated with the consumption of canned or preserved vegetables and fruits. 'Home-preserving' is carried out to a large extent, and this, especially as regards fruits and vegetables, is the real explanation of the relative frequency of the disease in that country.

Dickson (1917) carried out experiments to test the efficacy of the 'cold-pack' method then employed in home-preserving, where the filled jars were heated in a wash boiler to a temperature of 212°F. (100°C.) for 120 to 180 minutes. Dickson concluded that the 'cold-pack' was not efficient if the raw vegetables had been contaminated with the spores of *B. botulinus*.

Botulism apparently increased in the United States during the period 1910–22, but later showed a distinct tendency to decrease. Meyer holds the opinion that the decline in the number of outbreaks of single cases is attributable to energetic preventive measures, both educational and legal.

Since 1923, no cases of botulism have occurred in the United States attributable to commercially prepared foods. The following figures show that numbers of outbreaks and cases of botulism recorded in the United States Public Health Service reports during the past 5 years and attributable to 'home'-prepared foods:

Year	*Outbreaks*	*Cases*
1952	2	5
1953	7	10
1954	8	18
1955	5	14
1956	11	22

Botulism has been systematically studied by numerous scientific investigators in the United States and many brilliant contributions have been added to the literature on the subject by

Bengtson, Dack, Damon, Dickson, Dubovsky, Easton, Esty, Geiger, Jordan, Meyer, Ophüls, Tanner, Thom, Wilbur and others.

Dickson (1918), as a result of his intensive studies and investigations, compiled a clinical and experimental study on botulism. It may be of interest in passing to append some of his conclusions:

1. Botulism is endemic in the U.S. and is comparatively common in the Pacific Coast States.
2. It is not essentially a meat poison but may also occur in canned vegetables and fruits.
3. The methods which are usually employed in the home-canning of vegetables and fruits are unsafe.
4. All home-canned vegetables should be cooked before they are eaten.
5. Botulism is a frequent cause of the so-called 'limber-neck' of domestic fowl, and it may be responsible for certain types of paralysis of various kinds of domestic animals, including dogs.
6. The occurrence of limber-neck in domestic fowl, if it has developed after they have eaten refuse from the kitchen, may be an indication for the prophylactic administration of the botulinus antitoxin to all persons who have eaten the suspected foods.

In 1919 several spectacular outbreaks were caused by the consumption of factory-preserved olives, and for the first time the canning industry in America was confronted with the fact that *Cl. botulinum* was a real instead of merely a potential danger.

Impressed by the grave responsibility thrust upon the canning industry, the National Canners' Association, the Canners' League of California, and the California Olive Association proposed an investigation to determine the danger of botulism, how it arose and how it could be avoided and overcome. A commission (Geiger, Dickson, and Meyer) was formed in California and as a result of their researches *The Epidemiology of Botulism* was published in September 1922 by direction of the Surgeon-General.

Since 1929 the entire preserved foods industry of California has been controlled through legislation by the California State Department of Public Health, and revised tentative regulations covering sterilization of products are issued during the packing season of the year for the guidance of the industry.

Among the principal works in the United States containing matter relating to botulism are those by Jordan (1917, 1918, 1931), Dickson (1918), Bengtson (1924), Damon (1928), Meyer (1928, 1950), Tanner (1933, 1952), Strader (1939), Dack (1943, 1949, 1956). In addition, and as a result of experimental study of the disease by various workers, numerous articles have appeared from time to time in medical and scientific journals.

The presence of botulism in Russia (ichthyism) was reported in 1927 by Zlatogoroff and Soloviev. Twelve outbreaks occurred between 1881 and 1926 with 52 cases and 35 deaths. The disease appears to be endemic in the Republics.

Harmsen (1954) made a survey of the records and reports of instances of poisoning by botulinus toxin due to the consumption of fish or fish products and mentioned some of the conditions which favour the formation of the toxin, namely, humidity equal to at least 20 per cent water, anaerobic conditions sometimes localized in the less permeable parts of the food, a temperature of 25°–30°C.

In the great wars of 1914–18 and 1939–45 no cases of botulism were reported among the British or Allied troops, although the consumption of canned and preserved foods was enormous. This was doubtless due to rigorous inspection and supervision of supplies and to increased efficiency in the canning industry.

Dorendorf, however, recorded 5 cases of disease as occurring in the Germany army, and Bitter observed 3 outbreaks at Keil in 1918–19.

Legroux, Jéramec, and Levaditi (1946) gives statistics of botulism during the four years of German occupation in France. They estimated that there were at least 500 outbreaks and over 1,000 cases. The Pasteur Institute confirmed the diagnosis of 205 cases. The sources of infection in 163 cases were ham, either salted and smoked or pickled. In 30 cases the food preparations contained rabbit, duck, veal, goose, pork, fish, spinach, haricot beans, asparagus, peas, and *flageolets* (kidney beans); in 12 cases, pork, veal, or rabbit pies. Of the 295 outbreaks not bacteriologically investigated the source of the infection was known. For all 500 outbreaks, in no less than 93 per cent the origin was pig meat in one form or another. The foods causing deaths (15) were raw ham, preserved peas, preserved goose, preserved asparagus, a fresh tunny preparation, and food pies.

The toxins were Type A, causing 7 deaths, and Type B, with 8 deaths. These 15 deaths occurred in about 1,000 persons affected. Types A and B were about equally toxic. The authors call attention to this very low case mortality compared with outbreaks in United States and elsewhere. The gravity of the attack depended on the quantity of toxin ingested. All the fatal cases were those in which the food was taken cold. The very low fatality in those who were affected by slices of ham is probably explained by the small quantities eaten. Of over 900 persons affected from this source only one died.

Botulism in Great Britain

No authentic case of botulism were recorded in Great Britain until August 1922, when an outbreak (known as the Loch Maree tragedy) occurred at Loch Maree, Gairloch, in the Western Highlands of Scotland (See Chapter XXII). Eight persons were affected, and all died within a week after eating sandwiches made with wild duck paste. The outbreak formed the subject of a special report to the Scottish Board of Health by Gerald Leighton (1923). As a result of this lamentable tragedy the Ministry of Health made arrangements for a supply of botulinus antitoxic serum to be available at several centres in England and Wales and issued instructions for its use.

Leighton (1923) published his work on *Botulism and Food Preservation* and included a comprehensive study of the Loch Maree outbreak. This publication marked an epoch in the medical literature, as it was the first British work on the subject.

In the same year Humphrey Milford contributed an up-to-date review on the disease in the *Medical Science Extracts and Reviews*.

The Medical Research Council in 1929 published *A System of Bacteriology in Relation to Medicine* in which *Cl. botulinum* was dealt with by Hewlett, O'Brien, and Bulloch.

After the Loch Maree outbreak in 1922 there were no authentic cases until August 1935, when 3 deaths definitely due to botulism occurred in North London. The Chief Medical Officer, Ministry of Health (1935), reported:

> These fatal cases were all adult women, and there were two others, both male, in which the same intoxication was almost certainly a contributory cause of death . . . one man, who had partaken of the dish responsible for two of the fatal cases, recovered after presenting slight symptoms of botulism and being treated with botulinus antitoxin.

Later in this month another fatal case (a man) occurred in London. The findings at the autopsy were consistent with death from botulism.

Three cases of the disease occurred in 1944, but none were notified during 1945 and 1946. In 1947, however, there were 5 suspected cases with 1 death. After an interval of about 8 years, 2 cases (Mauritian students) of botulism (*Cl. botulinum* Type A) occurred in Battersea, London, during 1955, caused by the consumption of pickled fish and vegetables, prepared in and brought over from Mauritius. Both patients were treated with Type A antiserum and recovered.

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Chapter XVII

SYMPTOMATOLOGY OF BOTULISM

The symptoms of botulism which are manifested in man as a result of the gradual absorption of the toxin produced by *Cl. botulinum*, are of a peculiar and characteristic nature. They differ markedly from those observed in other types of food poisoning and vary in severity and duration in different outbreaks.

Van Ermengem (1897) remarks:

> The symptoms of botulism are so uniform and true to nature that for the recognition of the disease clinical appearances are alone sufficient. The picture is mainly made up of neuroparalytic disturbances of central origin. These processes find their expression in certain changes in the secretory functions of the digestive tract and in symmetric, generalised or localised motor paralyses caused by lesions seated in the ganglion cells of the bulbar and spinal nuclei.

In typical cases the symptoms develop in the same sequence, and the whole illness usually lasts from 36 hours to 5 days, but may be prolonged to a week. The majority terminate fatally, being due to cardiac or respiratory failure. In some instances, death may take place within 24 hours from time of onset. Few recoveries are recorded and in such cases convalescence is very prolonged.

Müller (1870) recorded that of 150 fatal cases, the majority died in from 4 to 8 days after the poisoning, and he added that few persons died who survived for more than 10 days.

In a series of 173 in America, 18 patients died within 48 hours after the food was eaten and one survived for 26 days; but 117 persons, or 67·7 per cent, died in from 3 to 6 days after ingesting the poison (Geiger, Dickson, and Meyer 1922).

The symptoms usually commence anything from 12 to 36 hours after ingestion of the toxic material, but may be delayed 2 to 3 days or even longer, considerable variation occurring in individual cases. Only rarely do they appear earlier than 12 hours. To illustrate this, Dickson (1918) says:

> In a series of over 200 cases a few occurred within 12 hours, 74 per cent. within 48 hours, and all but 8 cases within 4 days after the poisonous food was eaten ; 4 victims, however, first showed symptoms on the 5th day, 3 on the 6th day and 1 on the 8th day.

In a few instances the true intoxication manifestations may be preceded by gastro-intestinal disturbances, as observed in ordinary types of food poisoning. The earliest observers noted the gastro-intestinal disturbances, and Dann (1828) has suggested a division of the symptoms into two groups, i.e. the 'irritative' group and the 'paralytic' group; this was not generally accepted. Schlossberger (1852) and Müller (1870) pointed out that the irritative group of symptoms was frequently absent.

According to Geiger, Dickson, and Meyer (1922), approximately one-third of the cases of botulism have exhibited disturbances which usually come on early and apparently as a result of irritation of the alimentary tract by the spoiled food ingested. The remaining two-thirds show the typical symptoms of intoxication immediately following the incubation period.

In the classical outbreak of botulism at Loch Maree in Scotland (1922) intestinal disturbances were slight or absent altogether.

Regarding the incubation period, which is usually under 24 hours, Leighton (1923) remarks:

> The most unfortunate thing about the symptoms is that after the patients have taken the poison into their system, and while it is being absorbed and carried to the brain, there is a period extending over some hours during which no symptoms appear at all. The patient is quite unaware of what has taken place.

In botulism, the motor areas are very seldom affected. Paralysis is usually of the ascending type, gradually spreading upwards from the intestines until the medulla is reached.

The first signs are usually a peculiar feeling of lassitude, fatigue, headache, and dizziness, sometimes accompanied by progressive and definite weakness of the muscles of the arms and legs; vertigo is not uncommon. When gastro-intestinal disturbance is present there may be nausea and vomiting of a yellow colour, bitter taste, and irritating with a feeling of weight or actual pain in the region of the stomach. Diarrhoea may persist for a few hours or longer. As a rule intestinal disturbance is absent or of a transitory nature and of secondary importance. Persistent constipation (paralysis of the muscles of the wall of the intestines), which is a distinct feature of botulism, may be the initial symptom, or immediately follow the diarrhoeal stage and may or may not be accompanied by retention of the urine.

As the central nervous system becomes involved, which Bronfenbrenner and Schlesinger (1924) concluded was the result of the absorption of the toxin through the mucosa of the upper

intestinal canal—although according to Dickson (1918) instances have been recorded in which persons were poisoned by tasting very small amounts of the poisonous food and not swallowing any of the toxin—the visual disturbances begin to make their appearance, i.e. dimness or blurring of vision, double vision (diplopia), and early involvement of the third cranial nerve (ptosis), with drooping of the eyelids. The pupil of the eye increases in size (mydriasis) and involuntary oscillation of the eyeball (mystagmus) is not uncommon; fixation in the socket sometimes takes place. There is loss of reflex to light stimulation and finally complete loss of accommodation.

The ocular symptoms—impairment of the extrinsic and intrinsic muscles of the eye—are highly characteristic of the fatal form of intoxication and may be the first serious signs of the disease. Dickson (1918) points out that a fairly large number of cases of botulism are first seen by ophthalmologists and opticians. As intoxication proceeds, speech becomes difficult (husky voice), indistinct (dysphasia), and eventually loss of voice (aphonia) occurs. There is a sensation of suffocation and constriction in the throat, owing to paralysis of the pharyngeal and laryngeal muscles. The tongue becomes sluggish in movement, is heavily coated, increases in size, and may become paralysed. Swallowing is difficult (dysphagia) and attacks of strangling occur when an attempt is made to swallow food. During these attacks, regurgitation of fluids through the nose sometimes takes place. The muscles of the face and neck become affected, giving the patient a pale, dull, and mask-like expression.

In the early stages of the illness, restlessness, insomnia, irritability and sometimes hysterical attacks are observed as a result of the patient not being able to make himself or herself understood (except by writing) or to swallow. Secretory disturbances are most marked. In some cases there is unnatural dryness of the mouth, throat, and nose, the mucous membrane shrinks, shrivels, and desquamation may occur. In others, a thick glairy mucous exudes and stretches across the throat and pharynx, resulting in a croupy cough when efforts are made to free the mucous from the pharynx. Sweating may be absent, the skin on the palms of the hands and soles of the feet becoming dry and thick, but if present, is profuse and offensive. Partial deafness may ensue.

In mild attacks of botulism there is inco-ordination of the muscular movements of the arms and legs.

Extreme general muscular weakness is a marked feature of

the illness, the patient being unable to raise the head, arms, or legs.

Among the other characteristics are absence of sensory disturbances or pain, with consciousness and mentality unimpaired through the whole course of the disease. The temperature is subnormal ranging from 96° to 98°F. Pulse rate, which is comparatively slow in the early stages (50 to 60 per minute), later becomes rapid and as high as 100 to 150 per minute. This combination of rapid pulse rate and sub-normal temperature is a striking feature of the illness. In the early stages respiration is not interfered with but eventually breathing becomes irregular, shallow, rapid, and difficult. The normal blood-pressure and the skeletal muscle reflexes are intact. The patient becomes gradually weaker, the intercostal muscles are fatigued, the exhaustive strangling spells are more frequent, and finally death occurs from paralysis of the respiratory centres and cardiac failure, consciousness remaining almost to the end.

During the last few hours of the illness, broncho-pneumonia may supervene, which causes some fever with a consequent rise in temperature.

Cases have been recorded where coma has set in before death. According to Geiger, Dickson, and Meyer (1922):

> It has been frequently observed that the heart continued to beat for several hours after voluntary respiration had ceased, and cases are recorded when artificial respiration has maintained life for several hours after voluntary respiration had ceased. Usually there is terminal asphyxia with cyanosis, and occasionally the patient dies in a strangling spell. It is not uncommon that there may be apparent improvement in the general condition of the patient but that death results from insufflation broncho-pneumonia.

The diagnosis of botulism in post-mortem material is not easy. Neither toxin nor antitoxin can be detected in the blood, and secondary contamination of varying degree may render isolation of the organisms difficult. The liver must always be examined.

As to the cases which recover, it is generally recognized that the intoxication usually reaches its maximum in from 4 to 8 days, then it begins to subside. If, after 10 days, the patient survives, improvement usually follows, but convalescence is extremely slow and tedious. Recovery of speech and swallowing (strangling) takes place early. Owing to digestive troubles, the patient is thin and emaciated. Muscular weakness, vertigo, and constipation may persist for months and the visual disturbances are the last to clear up. Complete recovery, however, takes place, although it is some considerable time before the former condition of health is attained.

DIFFERENTIAL DIAGNOSIS BETWEEN BOTULISM AND OTHER KINDS OF FOOD POISONING

	Botulism (*Intoxication*)	*Salmonella* (*Infections*)	*Staphylococcus* (*Intoxication*)
Incubation period	6 hours to 8 days. Average 12 to 30 hours, sometimes delayed to 48 hours or longer.	5 to 72 hours. Average 6–12 hours, may be delayed.	½ to 6 hours. Average 2 to 4 hours.
Onset	Gradual.	Sudden.	Sudden.
Gastro-intestinal symptoms	Frequently absent; if present, slight and transitory.	Early and marked.	Common.
Vomiting	When gastro-intestinal disturbance is present.	Common.	Common.
Diarrhoea	Uncommon. Constipation early.	Usually severe. Offensive motions, which may later become watery.	Severe.
Abdominal pain	Usually absent.	Marked, often severe.	Present, often severe.
Muscular cramps	Absent.	Common.	May be present.
Temperature	Sub-normal 96°–98°F.	Variable elevated at first.	Variable.
Prostration	Gradual and often late.	Marked and early.	Acute.
Rashes	Absent.	Herpes common, also erythematous and urticarial rashes, followed by desquamation.	Absent.
Nervous system	Disturbances of vision, generally the first symptoms noticeable. Paralysis of accommodation, diplopia, mydriasis, ptosis and internal strabismus, aphonia, diuresis or anuria, and peresis of tongue.	Not often involved.	Not involved.
Duration of symptoms	Protracted and progressive. Convalescence slow.	Acute symptoms diminish rapidly after 48 hours to 3 days with exception of prostration.	5 to 6 hours or longer.
Mortality	Varies, 30 to 70 per cent or higher.	1 to 2 per cent	Nil or very rare.

Mortality

The mortality rate for botulism varies considerably in individual outbreaks, and figures ranging from 20 to 87 per cent have been recorded from time to time by various observers. In the Loch Maree outbreak in Scotland (1922) it reached 100 per cent. The rate in the United States is much higher than in Europe.

Regarding the death-rate among cases in the early history of botulism, Kerner (1820–2) records a series of 159 cases with 84 deaths—a mortality rate of 52·8 per cent.

In Schlossberger's (1852) series of 400 cases, there were 150 deaths, a rate of 37·5 per cent.

Dickson (1918) summarizes the early figures for cases and deaths in Germany from official sources obtained by Meyer, showing that the disease was still comparatively frequent in that country:

Date	*Cases*	*Fatal*
1793–1820	76	37
1820–1822	98	34
1822–1886	238	94

Since 1886 there have been about 800, about 200 of which were fatal.

In the United States, Geiger, Dickson, and Meyer (1922) collected data on 91 outbreaks up to 1922 with a total of 345 cases of which 213 were fatal, a case mortality rate of 61·7 per cent.

In the 11 recorded outbreaks in Colorado (1912–18), summarized by Hall and Gilbert (1929), the death-rate was 71·7 per cent.

In Germany the death-rate averaged 25 per cent (Mayer, 1913) and the United States 65 to 70 per cent (Burke *et al.*, 1921).

According to Zlatogoroff and Soloviev (1927) the fish-poisoning outbreaks of botulism in Russia were accompanied by a high mortality amounting to about 67·3 per cent.

The difference in the rate in America and Europe is probably attributable to the nature of the particular kinds of food consumed in the country concerned. The mortality rate for children is higher than for adults.

Apparently the earlier the symptoms of the disease, the higher the mortality rate. In America, Burke (1921) reported that among those showing symptoms in 24 hours, 84 per cent died; of those that developed symptoms in 72 hours, 55 per cent died, and of those alive after the eighth day, 20 per cent died.

Geiger, Dickson, and Meyer (1922) state:

> In a series of 246 cases where data were available and of which 173 resulted fatally, it was found that 147, or 85 per cent, of the fatal cases were persons in whom the onset of symptoms occurred within 48 hours after ingestion of the poison. In many outbreaks there is indication that the time of onset of symptoms is directly dependent upon the amount of poison ingested, and this observation shows that the severity of the illness and the mortality rate is also directly dependent upon the same factor.

From the above figure, it will be seen that the mortality rate for botulism is very high compared with other types of food poisoning.

Meyer (quoted by Jordan, 1931) points out the interesting fact that isolated cases and deaths from botulism occur most frequently in women who, as cooks and housewives, are likely to taste foods of their own preparation, which, from odour or appearance, they suspect.

Climatic Influence, Seasonal Prevalence, and Intoxication Rate

Botulism bears no relation to climate. As a rule the disease is usually associated with preserved foods, the consumption of which generally takes place in the winter months, when fresh food is not obtainable.

According to Dickson, Geiger, and Meyer (1922) more than half of all the outbreaks in California occurred between October to February in contrast with bacterial food infections, which are usually prevalent during the summer months.

The intoxication rate is very high (100 per cent: as a rule all who consume the toxic food become ill. Uneven distribution of the toxin in food is possible, but extremely rare.

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Chapter XVIII

CAUSATION OF BOTULISM

BOTULISM is an example of food poisoning due to bacterial products formed outside the human body. It is an intoxication and not an infection. In other words, for human botulism to result, the causative organism must multiply and produce its toxin in the food before it is consumed.

BACTERIOLOGY

Bacillus botulinus was described by Van Ermengem in 1896 as a very large slightly mobile anaerobic bacillus with rounded ends, 4–6 microns in length and 0·9–1·2 microns in breadth (a micron is 0·000039 of an inch). It sometimes occurred in pairs but rarely in filaments. The spores were terminal and somewhat wider than the bacillus, giving it a club-shaped appearance, and resisted ordinary stains. They were destroyed when exposed to 80°C. for 30 minutes. Spindle forms were sometimes observed. The bacillus was strictly saprophytic and would not produce its toxin in the animal body. It may truly be called a pathogenic saprophytic and placed in the same category as *Atropa belladonna* (deadly nightshade) and *Nux vomica*, among the green plants.

The organism, which had four to eight very fine flagella of wavy form, was gram-positive, but decolorized rapidly when treated with alcohol. Characteristically round, transparent yellowish-brown colonies were formed on glucose gelatine medium, containing granular bodies in motion. Gelatine was liquefied but milk was not changed or coagulated. Gas was formed in glucose broth or agar but in broths containing lactose or saccharose no gas was produced. A butyric acid (slightly rancid) odour was emitted during cultivation in various media. Van Ermengem believed that the amount of gas formation was an indication of the activity of the bacillus, especially in toxin production.

Media which gave an acid reaction to litmus or phenolphthalein prevented growth. Good growth, however, was obtained in a slightly alkaline medium containing 1·43 per cent of Na_2CO_3 incubated at a temperature between 20° and 30°C. (optimum temperature), producing a powerful toxin which, when fed to guinea-pigs and mice, proved very toxic. Rabbits, rats, pigeons,

dogs, hens, and cats withstood large doses of the toxin. Cats on subcutaneous injection showed all the characteristic symptoms of botulism. Monkeys (rhesus) were susceptible both to subcutaneous inoculation and to feeding. Frogs and fish were refractory. (After Bengtson, 1924).

Although a strict anaerobe, the bacillus may be cultivated under imperfect anaerobic conditions, if in symbiosis with certain aerobic bacteria, with the white *Sarcina* or with *B. subtilis* (Römer, 1900), and according to Harrass (1906) and Tarozzi (1905), will grow in freshly prepared bouillon conditions if a piece of sterile flesh or potato is placed at the bottom of the culture tube (Dickson, 1918).

A small amount of sodium chloride, 0·5 per cent, is necessary for the growth of the bacillus, but too much will inhibit development. Van Ermengem (1897) found that 2 per cent sodium chloride was deleterious to the growth of *B. botulinus* in bouillon Growth is stopped by 6 per cent sodium chloride; consequently meat pickled in brine containing more than 6 per cent will not become contaminated with the toxin.

Since Van Ermengem's discovery of *B. botulinus* several strains of the organism have been isolated from time to time in Europe. They differ in their characteristics and serological reactions from each other and from the original *B. botulinus* which of course has now died out. Damon (1928) states:

> Of the numerous strains now extant, that described as Lister No. 94, in the publications of the Medical Research Committee of Great Britain most nearly approaches the characteristics of Van Ermengem's culture and with this the American types may be compared.

A slight difference in regard to the degree of mobility may be noted. Van Ermengem states that his culture was very slightly motile. The Lister culture is described as actively motile (Bengtson, 1924).

Clostridium botulinum

Two main toxigenic types, designated 'A' and 'B' respectively, cause botulism in man. A and B were suggested by Burke (U.S.A. 1919), to distinguish the two groups. Type A, which is much commoner than Type B, has been isolated frequently from cases of botulism in the Pacific states, and Type B from cases occurring in the Eastern states and in Europe. In 23 outbreaks in America, 19 were due to Type A and only 4 to Type B (Topley and Wilson, 1936). A third Type C, was isolated by Bengtson (1922–3) from the

larvae of *Lucilia caesar*, one of the green-bottle flies, and is associated with a disease termed 'limberneck' of chicken, caused by the ingestion of the larvae (Wilkins and Dutcher, 1920, and confirmed by Graham and Boughton, 1923), and ducks in the United States and other countries (Gunnison and Coleman, 1932).

Further types were isolated in Australia and South Africa. An epizootic paralytic disease of cattle known as 'Midland cattle disease' exists in Tasmania, which was investigated by Seddon (1922). He isolated a bacillus which he named *B. parabotulinus*. This organism is considered to be a member of the Type C group.

Stubbs (1951) records that Type C developing in stagnant water (muddy ponds) can produce a toxin which when ingested by wild ducks or geese produces botulism which is frequently fatal.

Botija (1942) reports that botulism due to Type C toxin is a fairly common disease in horses, mules, and donkeys in the subtropical regions in Spain. The vector appears to be the farmyard cat, which, after eating putrefying organic matter containing *Cl. botulinum*, deposits its faeces on grain and other feeding stuffs and in this manner passes the toxin on to the solipeds. Tests show that the cat has a remarkable resistance to *Cl. botulinum* toxin. The faeces of cats on infected premises have been shown to contain large amounts of Type C toxin.

A fourth, Type D has been associated with a disease of horses, and cattle, *lamziekte*, in South Africa and isolated by Theiler and Robinson (1926–7), Robinson (1929), Gunnison and Meyer (1929).

A fifth, Type E, was demonstrated by Theiler and others (1926–7), Theiler (1928), in horses in South Africa.

Sterne and Wentzel (1952) report that botulism in South Africa is responsible for the death of thousands of cattle.

Meyer and Eddie (1951) in their paper on perspectives concerning botulism, discussed a new Type E, original strains of which were isolated in Russia from sturgeons. Outbreaks have since occurred in the United States and Canada due to Type E. The authors point out the close association of this type with fish.

Dolman and Chang (1952) record that about 14 Type E strains are known to have been isolated during the past 15 years in various countries: 6 in Russia, 4 in Canada, 3 in the United States, 1 in France; 12 of these were implicated in human botulism, and in all save one instance some form of fish was the vehicle. During their experiments they found that the more important contributory factors would seem to be the low thermal resistance of Type E spores and toxin, the ready production of Type E toxin in

uncooked fish, and the capacity of E strains to survive for several days in both sea and fresh water (see also Dolman 1957).

Regarding the types of *Cl. botulinum* isolated from botulism in animals, birds, etc., Topley and Wilson (1936) remark:

> Great confusion exists about the exact identity of the various organisms isolated. Because some of them differ in their toxin production from the classical *Cl. botulinum* A or B types, the names *Cl. parabotulinum*, *Cl. parabotulinum bovis*, or *Cl. parabotulinum equi* have been suggested, and the disease caused by them has been termed parabotulism. This is not the place to discuss bacteriological nomenclature, but we are in entire agreement with Weinberg and Ginsbourg (1927) that for the moment these organisms should be regarded as varieties of *Cl. botulinum* and referred to as *Cl. botulinum*, Type C, D or E. Their relationship to each other and to the two classical types is very uncertain, and apart from slight differences in the nature of the toxin produced, there seems to be nothing that would justify their elevation to specific rank.

Bengtson (1924) studied the serological reactions of a number of strains of *Cl. botulinum* concerned in the causation of botulism and grouped them as Types A, B, and C. All the strains are anaerobes and suitable conditions are necessary for their cultivation in media. After a comparative study of over 100 strains of the bacillus, Meyer (1922) expressed the opinion that growth and toxin production takes place best at 35° to 37°C. One tenth of the amount of oxygen present in the atmosphere will inhibit the growth of all the types (Dack and Baumgartner, 1928). Types A and B can grow approximately 7·5 per cent of normal atmospheric O_2, but Type C will develop only when the amount of oxygen is less than 3 per cent of the atmospheric O_2 (Jordan, 1931).

It has been found that Types A and B can be distinguished by experimental feeding to chickens, the birds being susceptible to type A only, death occurring in from 18 to 24 hours. Suggestions have been made that this test might prove useful for identifying the strains responsible for cases of human botulism.

Type A, which is a large, thick, motile bacillus, with rounded ends, occurring singly or in pairs or chains, differs in some of its characteristics from Type B. Its subterminal spores are very resistant to heat and under favourable circumstances remain dormant for quite long periods before germinating. Type B more closely resembles *B. botulinus*, described by Van Ermengem, as it is easily killed by boiling. Types A and B are distinguishable by the fact that the toxin of one is not neutralized by the anti-toxin of the other.

Type C is a gram-positive, anaerobic, spore-bearing, slightly motile bacillus. The organism, however, is longer and more slender than A or B strains and occurs singly or in pairs or chains. Damon (1928) remarks:

> The organisms belonging to this type differ distinctly in their cultural characters from the other American strains, but produce a symptom complex in animals that is indistinguishable from that produced by the Type A and B organisms.

The table on p. 318 shows the cultural characters differentiating *Cl. botulinum* types A and B from Type C and *parabotulinus* of Seddon.

With regard to carbohydrate fermentation, according to Bengtson (1924) A and B strains ferment dextrose, levulose, maltose, glycerol, and dextrin, but fail to ferment galactose and inositol. Type C strains, however, ferment galactose and inositol. Salicin is fermented vigorously by most A and B strains. Indol is not produced by any known strain (Norton and Sawyer, 1921).

Occurrence and Distribution in Nature

Available evidence shows that *Cl. botulinum* is found all over the world, its natural habitat being the surface layers of virgin soil; it is present, too, in cultivated and other soils. The longer the soil has been in cultivation, the less common is the organism.

The spores of the bacillus may gain access to vegetables, fruits, and other cultivated produce and be transported by insects and even swallowed by cattle and other animals.

History relates that Van Ermengem (1897) after his discovery of *B. botulinus*, endeavoured to prove its existence in nature. He examined 52 samples, which included excreta of domestic animals, intestinal contents of fishes, and specimens of soil, mud and manure, but without success. In Berlin, Kempner and Pollack (1897) found a bacillus in a pig's faeces. Van Ermengem examined the organism, which resembled the original *B. botulinus*, but more closely corresponded to the Darmstadt (1904) type of organism. Dickson (1917) examined the contents of the intestines of 250 grain-fed pigs from San Francisco but failed to isolate the organism.

The majority of the investigations on the occurrence and distribution in nature of *Cl. botulinum* has been carried out in America. Burke (1919) studied the subject in California and found that *Cl. botulinum* was widely distributed in nature and was present in garden soils, thus making it possible for fruits, vegetables, etc., to be contaminated by the spores of the organism.

Burke concluded that the bacillus existed near human dwellings and was spread by spiders and other insects, but that the organism was not necessarily associated with the faeces of warm-blooded animals.

The question arose as to whether *Cl. botulinum* was an intestinal saprophyte and consequently occurred in cultivated regions, or whether it belonged to the ordinary flora of the soil and could increase under natural conditions.

(After Graham and Boughton)

	Clostridium botulinum Types A and B	*Clostridium botulinum* Type C or *para-botulinus* (Seddon)
Glucose agar	Gas	No gas
,, ,, .	Disc colonies	Branching colonies
,, broth	Even cloudiness	Flocculent growth
,, ,, .	Acid and gas	Acid
Meat mash .	Very fine gas bubbles on surface	Gas bubbles large and along sides of tube
Milk . .	No change	Acid
Motility .	Motile under cover glass	Non-motile under cover glass
Spores .	Resistant to heat	Non-resistant to heat

The positive results obtained from the extensive investigations by Meyer and Dubovsky and their colleagues (1922), who examined 1,533 soils of all descriptions, manure, vegetables, etc., collected in the United States, Canada, Belgium, Denmark, England (many counties), Holland, Switzerland, Hawaiian Islands, China and other countries, indicate that *Cl. botulinum* is a natural inhabitant of the soil and of widespread distribution. It also shows that the organism is by no means evenly distributed and is commoner in virgin soil and pasture than in cultivated soil. Schoenholz and Meyer, 1922; Hall and Peterson, 1924; Damon and Payabal, 1926.

Regarding the types of organisms isolated, Type A was found chiefly in virgin soil. In the 335 samples examined 59 showed Type A and 22 Type B. In the 274 specimens of cultivated soil 18 showed Type A and 16 Type B, and in the 51 pasture samples 3 showed Type A and 11 Type B.

In the European soils Type B was predominant. Of the 64 specimens collected from different counties in England, 5 samples showed Type B. (Meyer and Dubovsky, 1922.)

Type C (Graham and Boughton, 1923–4) was found in soils collected from chicken-runs and stables where outbreaks of limberneck or botulism in horses had occurred.

As to the distribution of Types A and B, Topley and Wilson (1936) remark:

> Meyer and Dubovsky's results have not as yet received general confirmation; some of their conclusions may have to be modified (Geiger and Benson, 1923; Bachmann and Haynes, 1924), and more work must be carried out before the relationship of the two types to environmental conditions can be definitely determined.

Leighton and Buxton (1928) made investigations into the distribution of *Cl. botulinum* in Scottish soils, and examined 160 samples from cultivated gardens, ploughed fields, pasture land, and uncultivated waste moorland. Positive results were obtained from 4 of the samples; pasture land 3, ploughed land 1. Two were Type A, 1 Type B, and 1 Type A and B.

Parry (1947) collected and made bacteriological examinations for *Cl. botulinum* of 283 samples of soil (cultivated and virgin) and also dust, from the five central counties of New York State. *Cl. botulinum* Types A and B was found to be distributed widely in the central area. Thirty-three, or 11·7 per cent of the total samples yielded toxic cultures of *Cl. botulinum.* Twenty-six of these were Type A; 5 or 15·1 per cent Type B; and 2 or 6·6 per cent yielded a mixed toxin of Types A and B.

Cl. botulinum has been occasionally isolated from the excreta of horses, pigs, and cattle, which feed on soil produce (Burke, 1919; Tanner and Dack, 1922; Easton and Meyer, 1924). Meyer (1924) concluded that the evidence secured from an examination of 95 manure specimens strongly indicates that animal excreta contributes relatively little to the pollution of the soil with *Cl. botulinum.*

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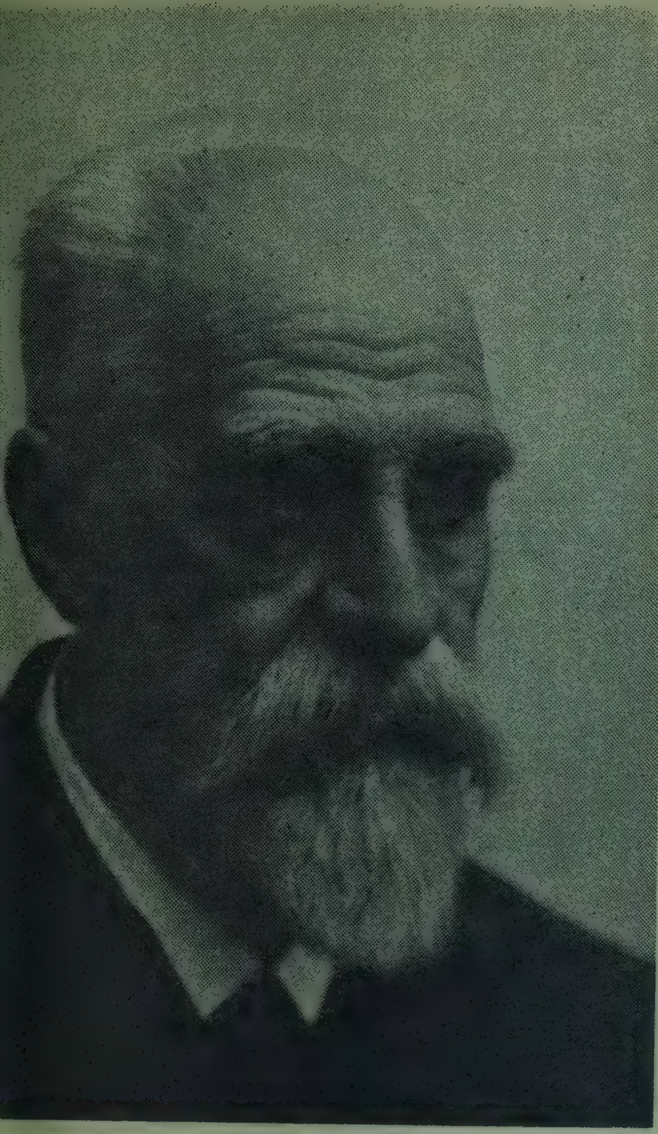
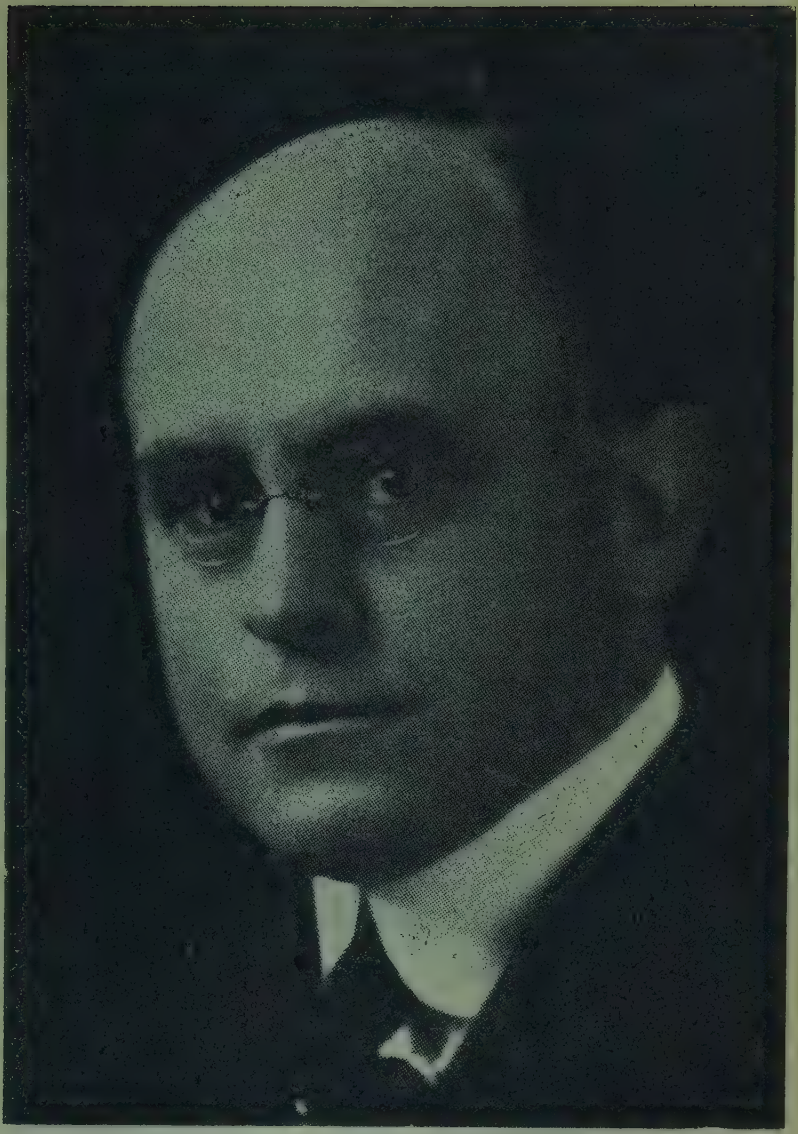
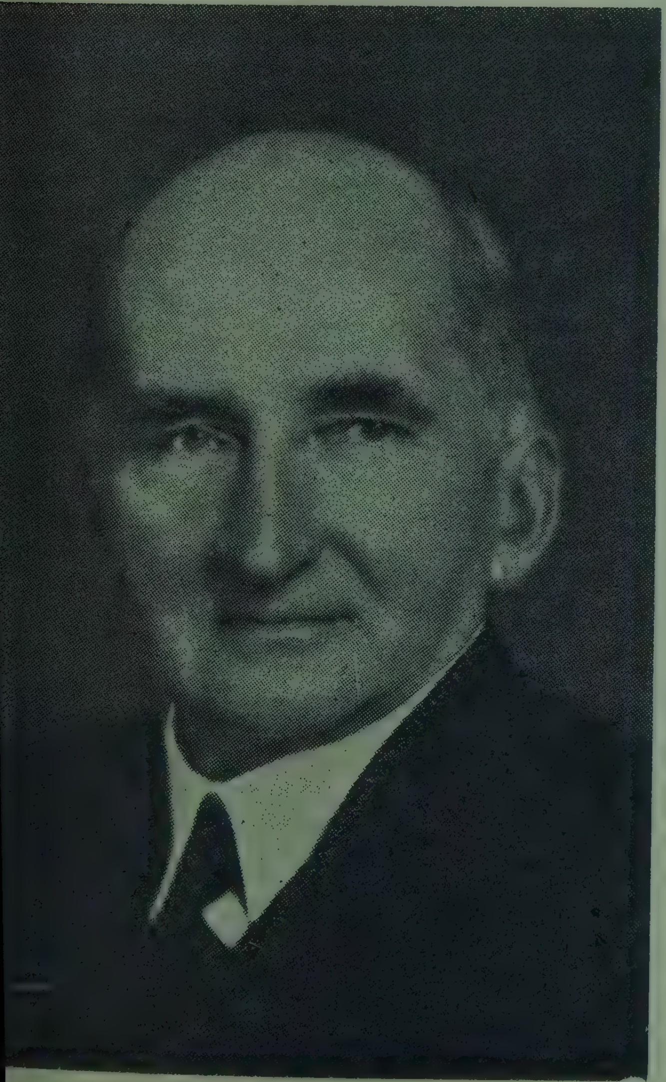


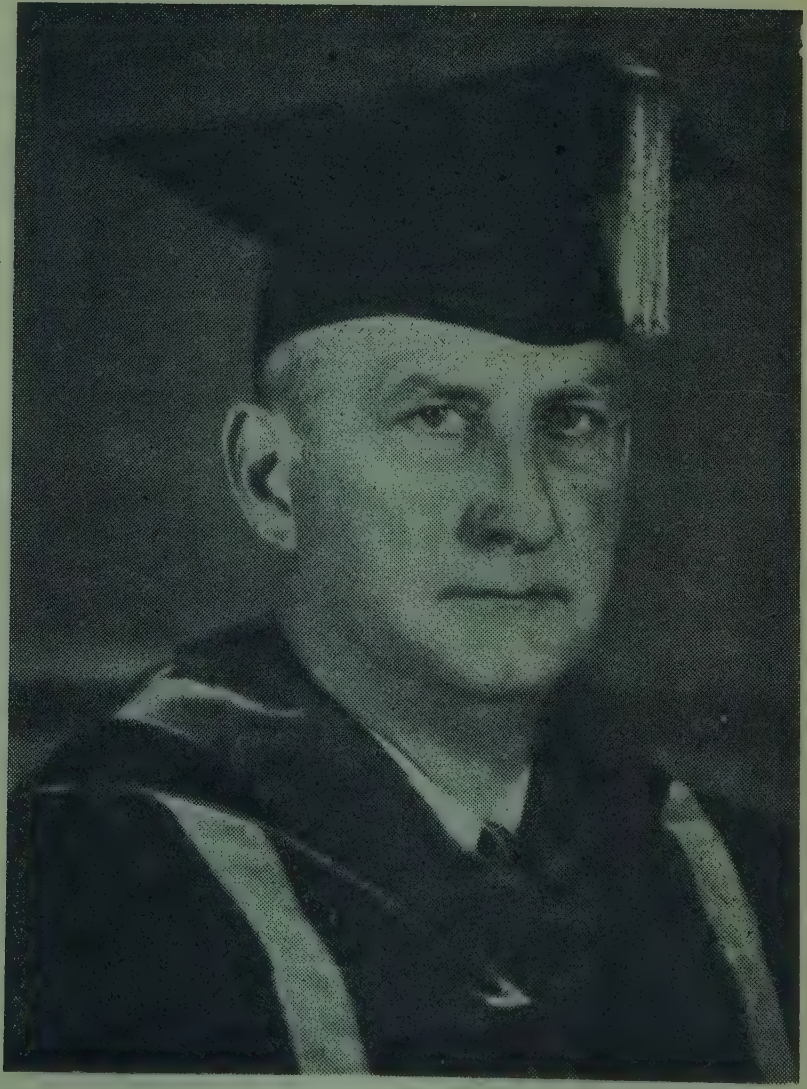
Professor Emilé P. M. Van Ermengem



(*b*) Dr. Ernest C. Dickson



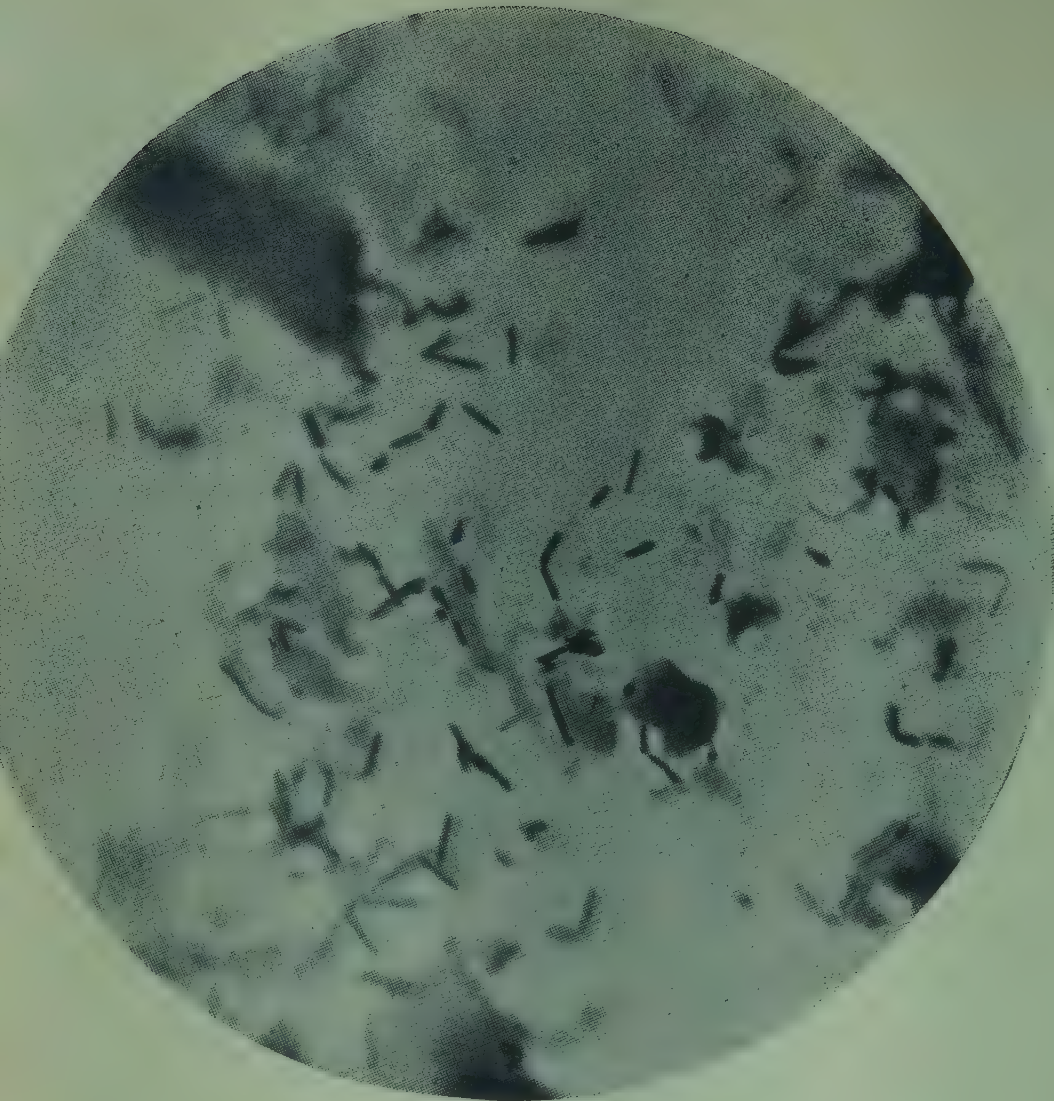
(*c*) Dr. Gerald R. Leighton



(*d*) Dr. J. G. Geiger

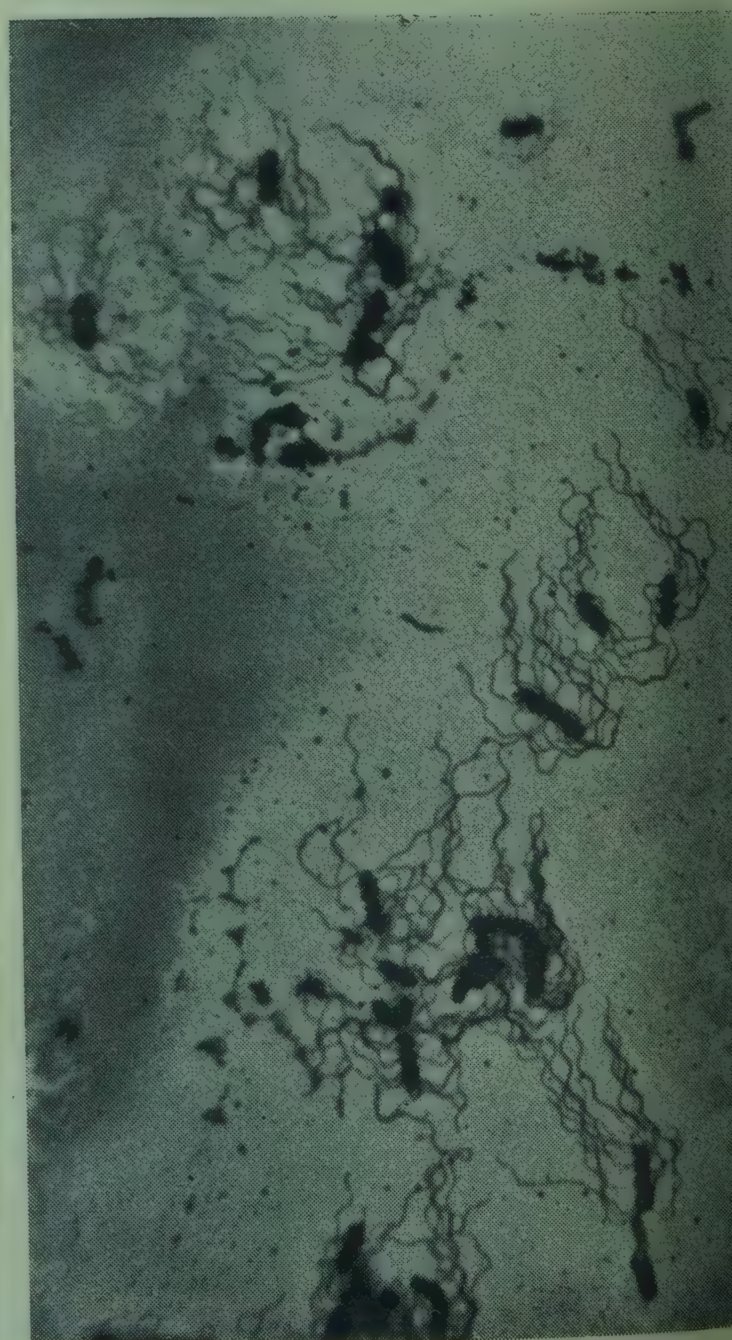
PLATE 33

Plate 34

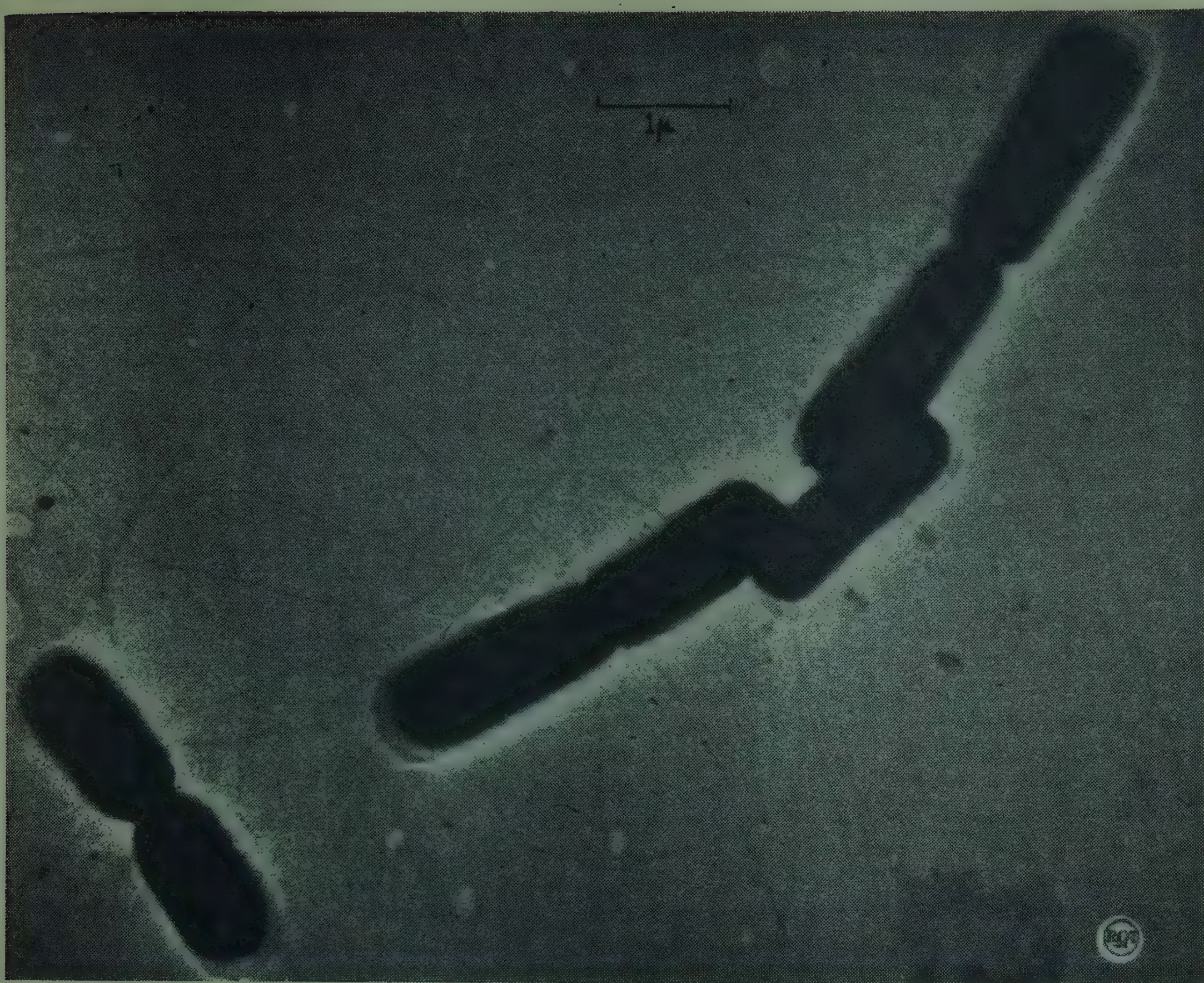


(*a*) *Cl. botulinum*, Type A

(*b*) *Cl. botulinum*, Type B



(*c*) *Cl. botulinum*, Type C



(*a*) Electron micrograph, *Cl. botulinum*

(*b*) Professor Karl F. Meyer, M.D.

PLATE 35

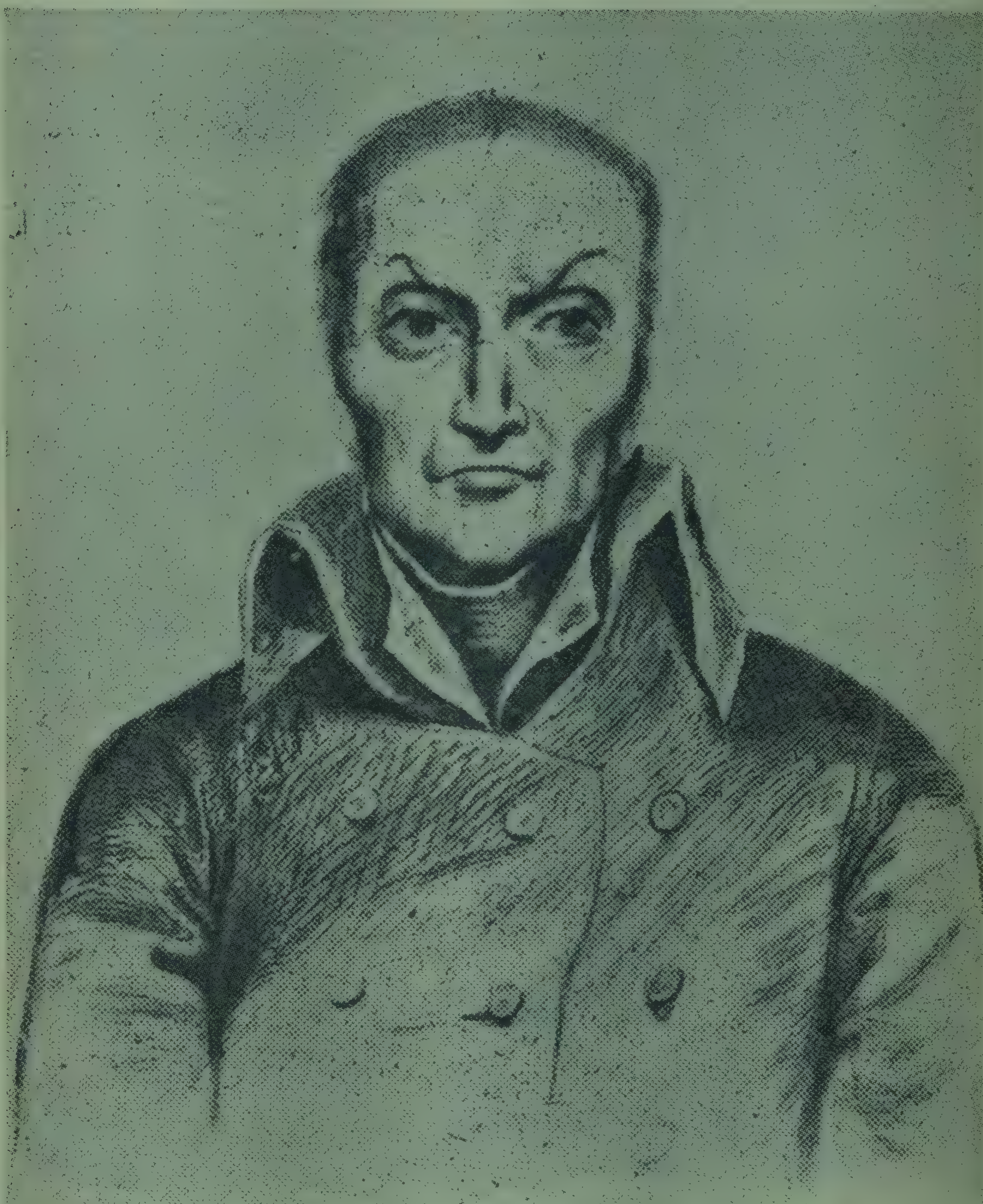
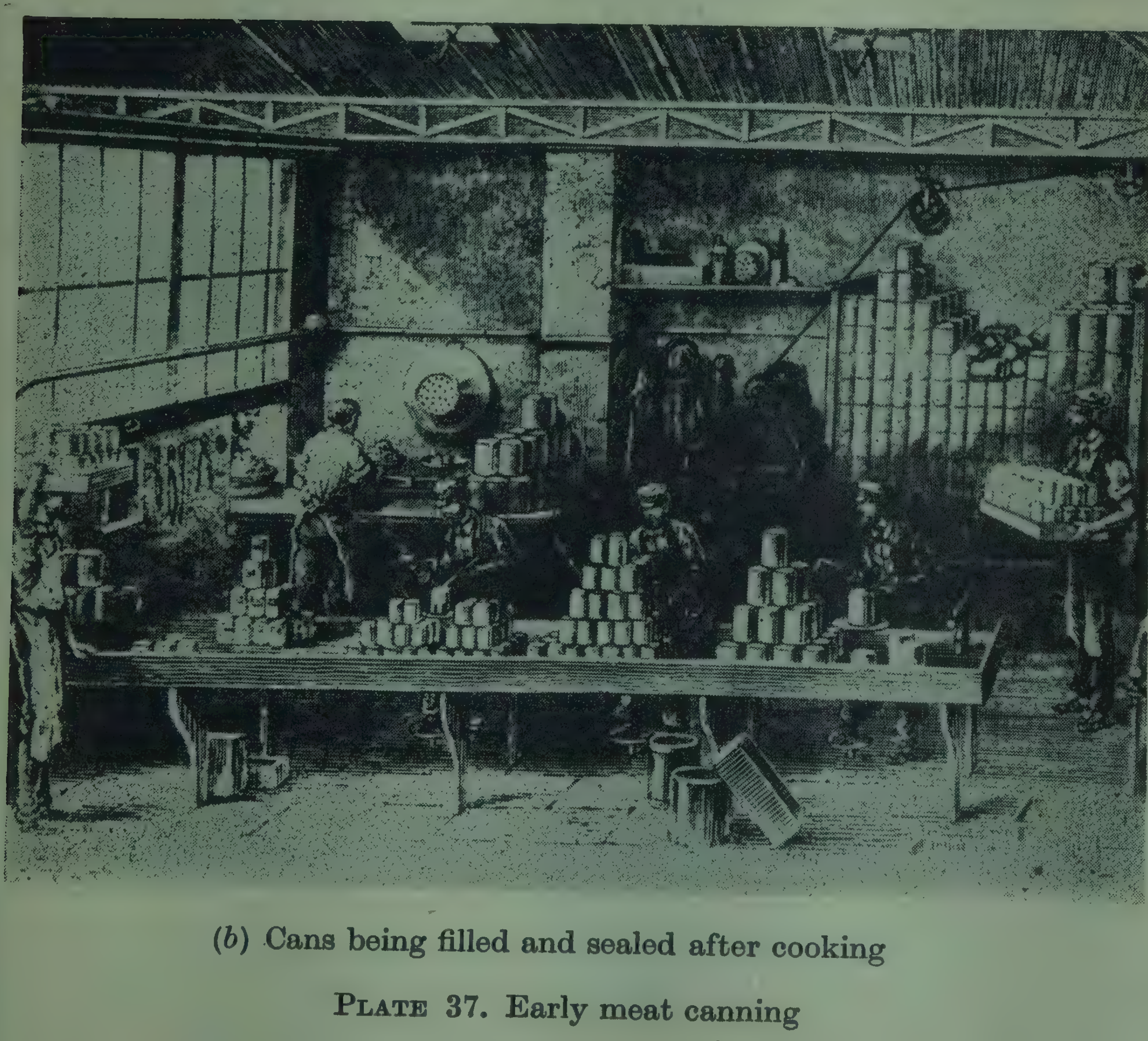


PLATE 36. Nicholas Appert, 1752-1840

(*a*) Preparation room in a canning establishment



(*b*) Cans being filled and sealed after cooking

PLATE 37. Early meat canning

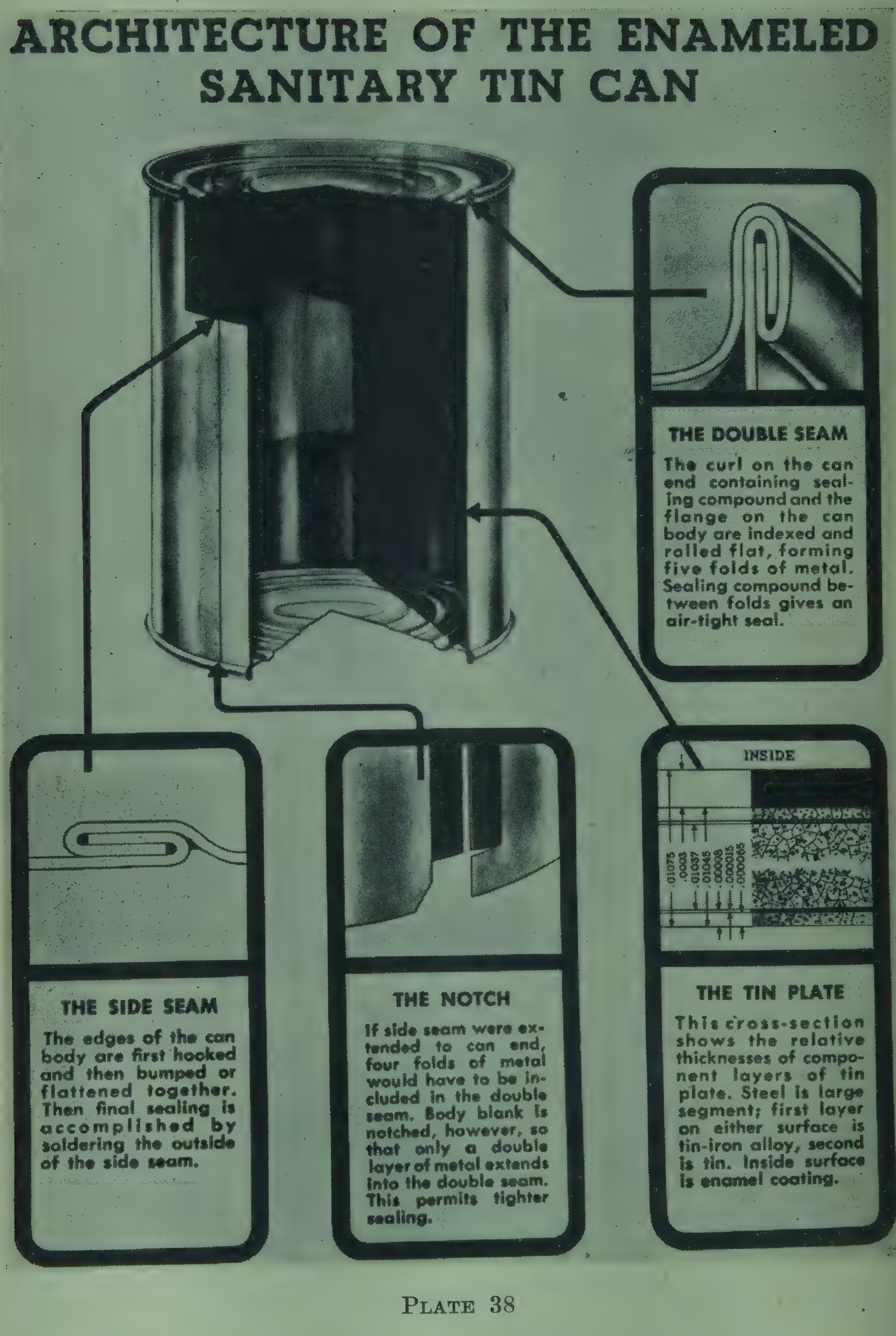

ARCHITECTURE OF THE ENAMELED SANITARY TIN CAN
THE DOUBLE SEAM
The curl on the can end containing sealing compound and the flange on the can body are indexed and rolled flat, forming five folds of metal. Sealing compound between folds gives an air-tight seal.
INSIDE
.01075
.0003
.01037
.01045
.00008
.000015
.000065
THE SIDE SEAM
The edges of the can body are first hooked and then bumped or flattened together. Then final sealing is accomplished by soldering the outside of the side seam.
THE NOTCH
If side seam were extended to can end, four folds of metal would have to be included in the double seam. Body blank is notched, however, so that only a double layer of metal extends into the double seam. This permits tighter sealing.
THE TIN PLATE
This cross-section shows the relative thicknesses of component layers of tin plate. Steel is large segment; first layer on either surface is tin-iron alloy, second is tin. Inside surface is enamel coating.

PLATE 38

(*a*) Hole-and-cap can

(*b*) Sanitary tin can

Dents on a can do not necessarily indicate spoilage of contents

Rust on a can rarely indicates spoilage of contents

(*c*) Dents and rust on cans

PLATE 39

Plate 40. Ida Bengtson, Ph.D.

Chapter XIX

SPORES OF *CL. BOTULINUM*

Spore Formation

If the bacillus is cultivated in a suitable medium at the optimum temperature, spores will usually form and germinate, but in unfavourable conditions their formation is delayed or even prevented altogether.

Regarding the function of spores, it may be of interest to mention that in 1931 Jordan wrote:

> Physiologically the spore is usually considered as a resting stage, serving to tide the species over a period of dryness, famine or unsuitable temperature, and to preserve alive in a hostile environment a sufficient number of individuals until such time as favourable conditions recur. In this view the spore stage is physiologically analogous to the periods of hibernation or estivation among higher forms of life, and the living matter of the spore may remain dormant for years or even decades.

The dormancy of spores has received much attention owing to its importance in the canning industry and thermal death-time determinations.

According to Dickson and others (1925), the spores of *Cl. botulinum* retain their vitality for long periods if protected from the action of light and air.

Resistance to Heat

This has been carefully studied experimentally in America by many observers. The destruction of the spores of *Cl. botulinum*, which can withstand high temperatures for long periods and boiling water for ½ to 22 hours (Bigelow and Esty, 1920; Weiss, 1921; Esty and Meyer, 1922; Tanner and Twohey, 1926), and 120°C. for 20 minutes, is an important consideration in the prevention of human botulism.

The spores of some strains of *Cl. botulinum* are more distinctly heat-resistant than those of any other anaerobes. The fact that delayed germination of the spores sometimes takes place, even after they have been subjected to a comparatively high temperature, adds to the difficulty of determining their heat-resistance. Jordan (1931) remarks:

> Indeed, if the germination of the spores be inhibited so that growth and consequent toxin production are prevented, botulism cannot occur.

It has been recognized that in canning and preserving foods, acidity (the intensity factor of acidity, not the percentage of acid present) is one of the chief factors affecting time and temperature for the destruction of the spores of *Cl. botulinum*. In other words, a close relationship exists between their heat-resistance and the hydrogen-ion concentration which is expressed in terms of *p*H value. Products with a *p*H value below 4·5 are not usually subject to spoilage when packed under satisfactory sanitary conditions. The higher the hydrogen-ion concentration the shorter the time required for the destruction of the spores. The hydrogen-ion concentration necessary, however, to inhibit their development varies according to the nature of the acid and the specific strain Meyer (1928). In the medium in which they are heated, the spores germinate freely at a *p*H value of 6·0 to 7·2. (Note that pure water which is neutral (neither acid nor alkaline) has a *p*H value of 7. A large number of foods are more or less acid. The more acid the food, the lower its *p*H value—the more alkaline it is, the higher the *p*H value.

Esty and Meyer (1922), who carried out extensive investigations, found that at *p*H 7·0 the spores were killed in 330 minutes at 100°C.; at *p*H 5·05 in 45 minutes, and at *p*H 3·7 in 10 minutes. The smaller the number of the spores in the food the shorter is the time necessary to destroy them (Bigelow and Esty (1920)).

Esty (1923) found that the spores of Type A were more resistant than those of Type B. Type C strains form less resistant spores. After much intensive experimental work, Esty came to the conclusion that all the spores of *Cl. botulinum* would be destroyed at the following times and temperatures:

	minutes
100°C.	360
105°C.	120
110°C.	36
115°C.	12
120°C.	4

There is considerable variation in the heat-resisting properties of spores of different strains, even with spores of the same strain and even under controlled experimental conditions. Tanner (1933) remarks:

> The heat-resistance of spores in nature is probably quite different from that of spores under artificial conditions of the laboratory. Practically all our data on heat-resistance have been secured on spores grown in the laboratory. In many cases the menstruum in which the spores were suspended is an unusual one and not like those in food.

Pilcher recently recorded the following thermal death-times for spores of *Cl. botulinum:*

	minutes
126·7°C. (260°F.)	0·78
123·9°C. (255°F.)	1·45
118·3°C. (245°F.)	2·78
121·1°C. (250°F.)	5·27
115·6°C. (240°F.)	10·00
110·0°C. (230°F.)	36·00
104·4°C. (220°F.)	150·00
100·0°C. (212°F.)	330·00

The spores of *Cl. botulinum* are not killed by weak acids or by fairly strong brine concentrations. Food containing salt (sodium chloride) lowers thermal resistance, which decreases with increasing concentration of the salt (Weiss, 1921).

According to Esty and Meyer (1922), no decrease in their resistance to heat was noticed until 8 per cent salt solution was reached. Spores are not destroyed by prolonged exposure to cold; they can survive freezing at –16°C. (–3·2°F.) for 14 months, as shown by Wallace and Park (1933).

James (1933) in a study of the effects of freezing on the spores and on the toxin of *Cl. botulinum*, states:

> Dried spores of Cl. botulinum Type B (Onion strain) suspended in buffer solution of *p*H 6·9 in pea juice *p*H 7·1 were not killed or materially reduced in number by either slow or quick freezing. Botulinus toxin frozen and defrosted 15 times was not reduced in strength.

Keppie (1951) as a result of investigations concluded that the spores of *Cl. botulinum* present in spoiled foodstuffs are not of importance compared with the toxin produced by the initial growth of the contaminating organisms.

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Chapter XX

TOXIN AND ANTITOXIN

Toxin of *Cl. botulinum*

The extremely poisonous substance produced during the growth of *Cl. botulinum* (strains A, B, and C) in a suitable culture medium under strict anaerobic conditions and at a proper temperature, is a powerful filterable bacterial exotoxin possessing certain physical and chemical properties. Its production is more uniform when glucose is present in the nutrient medium.

The virulence of the poison varies greatly; it depends upon the strain of the organism, medium temperature, and conditions of anaerobiosis (Dickson, 1918). Not all strains form toxin, but the majority do (Bengtson, 1922; Starin, 1924).

Meyer (1929) found that an incubation period of 10 days at a temperature of 35° to 37°C. produces a toxin of the highest potency. It may, however, develop in small quantities below 20°C. and up to 34°C.

Dickson (1918) during his investigations and experiments found that the strongest toxin was produced in pork and beef infusion, but virulent poisons were also excreted in media prepared from string beans, green peas, and green corn, respectively. Much less virulent toxins were obtained in media prepared from asparagus, artichokes, peaches, apricots and crushed apricot stones.

The toxin is insoluble in alcohol, ether, or chloroform. Cold may act as a deterrent to its development in food, but as soon as the latter (containing the spores) is warmed up, toxin formation may occur.

Wallace and Park (1933) showed that no decrease in potency occurred when the poison was stored at –79°C. (–110·2°F.) for 2 months or at –16°C. (–3·2°F.) for 14 months.

Under experimental conditions the toxin is destroyed by the prolonged action of direct sunlight, diffuse daylight, and air (Schoenholz and Meyer, 1924; Bengtson, 1924), but if kept sealed and in the dark, it retains its potency for long periods. Putrefaction has no effect on its virulence if access of air is prevented (Dickson, 1918).

It is resistant to acids. Van Ermengem (1896) observed that tartaric and lactic acids in the proportion of 1 to 3 per cent and hydrochloric acid in the proportion of 0·5 to 1 per cent did not lower the toxicity of a filtrate after incubation from 24 to 36 hours at 35°C.

Bronfenbrenner and Schlesinger (1924) found that the poison resisted acidity equal to that of the stomach for 24 hours at 37°C. and noticed that the potency was increased by acidification.

Alkalis exert a powerful effect upon the toxin as observed by Van Ermengem (1896). This was confirmed by Landmann (1904) and by Bronfenbrenner and Schlesinger (1924).

The poisonous property of the toxin is such that the fatal dose for an adult man, calculated on the basis of animal experiments, might be as small as $\frac{1}{100}$ mg. or even less.

The three types produce toxins of different potency, the ratio of lethal dose for Types A, B, and C, respectively, being approximately 1 : 50 : 125 (Bengtson, 1924).

As illustrating its highly poisonous and fatal nature, Dickson (1918) records the case of a woman who died after nibbling a portion of a pod of spoiled string beans and of another who succumbed after tasting a small spoonful of spoiled corn. He remarks:

> It is known that the sub-lingual mucosa permits fairly rapid absorption, and it is possible that the toxin may be absorbed in fatal quantities by this route.

The poison after ingestion resists the gastric secretions and is absorbed by the mucosa of the stomach and upper intestine without undergoing alterations and gives rise to the disease.

The action on human beings is obscure, but its principal effect is upon the motor nerve endings. Dickson (1918) states:

> It is possible that the toxin acts, as does belladonna, upon the terminal end-plates of certain nerves, and the close resemblance between the effects of the botulinus toxin and those of the administration of belladonna suggest that this may be true.

Guyton and MacDonald (1947) concluded that there was no evidence that the botulinum toxin acted on the nerve trunks, but the site of action took place in the terminal nerve fibrils or as suggested by Bishop and Bronfenbrenner (1936) at the myoneural junction. The toxin also causes peripheral circulatory failure.

Minute quantities of the toxin are fatal when introduced into suitable experimental animals (rabbits, monkeys, cats, pigeons, etc.) either by subcutaneous, intraperitoneal, or intravenous injection, or by feeding. As little as 0·0003 to 0·001 c.c. of a broth

culture may kill a rabbit. The poison produces all the characteristic symptoms of botulism (Savage, 1920).

In laboratory experiments it has been possible to obtain a toxin of which 0·000001 c.c. will kill a 250 gm. guinea-pig in from 3 to 4 days (Brieger and Kempner, 1897). According to Guyton and MacDonald (1947), a pure crystalline preparation of the toxin would probably prove fatal to man in a dose of a quarter of a millionth of a gramme.

Researches by Geiger (1924) indicate that it is possible to poison experimental animals by absorption of the toxin through lacerations in the gums or abrasions in the skin or from uninjured mucous surfaces.

Therefore extreme care should always be taken in handling suspected contaminated packs of food, though it is fully recognised and appreciated that from the epidemiology of the majority of the botulism outbreaks that have occurred, this is a remote possibility.

It is true that there is no proved record of the poisoning of human beings except when the toxin is ingested.

Unlike the spores of *Cl. botulinum*, the toxin is quickly destroyed by heat, but the time taken for destruction varies with the strain of the bacillus and the temperature. According to Jordan (1931),

exposure for from 6 to 10 minutes at 80°C. is sufficient to inactivate the toxin produced by most Type A strains; the Type B toxin needs a somewhat longer exposure (15 minutes), and the Type C toxin is still more resistant (up to 30 minutes at 80°C.).

Abrams, Kegeles, and Hottle (1946) prepared a crystalline globulin possessing the immunological and biological properties of the toxin of *Cl. botulinum* Type A. The crystalline toxin had the following properties: (1) uniform needle-shaped crystals; (2) electrophorectic homogeneity; (3) a single sharp ultra-violet absorption band 278 mμ.; (4) nitrogen 14·1 per cent (V) 220×10^6 mouse m.l.d. per mgm. N_2. The toxoid was highly antigenic to mice.

Botulinum Antitoxin

The toxins of the various strains of *Cl. botulinum* gives rise, when suitably injected, to specific antitoxins. The specific antitoxin of one type, however, will not protect against the toxin of another type. This is contrary to the situation observed in the case of other toxicogenic organisms (Damon, 1928).

Jordan (1931) states:

> It is a remarkable fact that the characteristic physiological action of the three toxins (A, B and C) seems identical, that no marked cultural difference between the types has yet been made out and that the agglutination reaction shows intergrading of the types as well as differences within each group. The only basis on which a type distinction seems warranted is the specific nature of the antitoxin.

Early experimental work on antitoxin production and its therapeutic properties was carried out by Kempner and Pollack (1897). They also demonstrated the curative value of the serum and found that when the serum was subcutaneously injected into guinea-pigs after the appearance of symptoms of intoxication, some of the animals recovered, in others death did not supervene for weeks or months. Thus a certain curative value for the antitoxin was proved.

The antitoxin sera can be prepared by the injection of goats (Kempner, 1897, Forssman, 1905) or horses (Leuchs, 1910) or rabbits (Nevin, 1921), with each type of toxin.

Damon (1928) points out:

> The curative value of antitoxin in human cases has not been definitely established, but there is some evidence that it may be employed effectively in prophylactic doses. The curative property depends on the elapsed time since the ingestion of the toxic food and the amount of toxin consumed.

Favourable results in human cases have been reported (McCasky, 1919; Geiger, 1920). According to Topley and Wilson (1936), 'large doses, 50 c.c. or more, of polyvalent serum, or of monotypical serum if the type of the intoxicating organism is known, should be given intravenously every day till the patient recovers, or all hope is abandoned. A prophylactic dose of 10 c.c. should be given intramuscularly to all who have partaken of the poisonous food and who have not yet developed symptoms of the disease.'

Meyer (1953) recommends that early treatment with bivalent (Types A and B) antitoxin serum in a dose not less than 10,000 units every 18 to 24 hours. It should be liberally dilated with a 5 to 10 per cent solution of dextrose and injected intravenously.

The Ministry of Health has made arrangements for a suitable supply of botulinus antitoxic serum to be available for medical officers of health and medical men in case of need at several centres in England and Wales.

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Chapter XXI

FOODS ASSOCIATED WITH OUTBREAKS OF BOTULISM

History relates that the incriminated foods in the majority of the earlier cases of so-called 'sausage poisoning' were of animal origin, i.e. blood or liver sausage, blood puddings and other 'made-up' meat foodstuffs. The ingredients used in the preparation of these articles consisted chiefly of liver, sheeps' brains and plucks, tongues, veal, pork, calf or goats' blood, and fats of various kinds. They were packed in skins or casings (stomach or large intestines), which were easily procurable and inexpensive, but on account of their nature and size, difficult to smoke satisfactorily. Being uncooked or only partially so, they did not resist putrefaction to any extent; moreover, according to historical records, were sold to the poorer classes and sometimes eaten raw. Small meat sausages were also manufactured and apparently prepared and smoked or cooked by skilled workmen under improved sanitary conditions and more expensive.

It is an interesting and significant fact that Kerner (1820) noted that when the sausage casings were incompletely filled they did not become toxic, and he concluded that exclusion of air was necessary for the development of the poison which caused the characteristic symptoms of the illness. Kerner also observed that the poisonous food had a peculiar odour, differing from that of putrefaction.

In recent outbreaks of botulism in Central Europe, other foods of animal origin were implicated. These were in more or less spoiled condition, probably due to being partly smoked—incomplete impregnation with the antiseptic substances of wood-smoke or inadequately home-pickled or insufficiently cooked. Among these foodstuffs were smoked, pickled or salted hams or fish, pork brawn, preserved meats, game *pâtés*, potted or smoked goose or duck, etc.

It has been suggested that in the case of hams the contamination was introduced through the bone marrow. Toxic portions have often been found in the deeper parts, which is favourable for the growth of the spores of *B. botulinus*. The only German outbreak which was definitely traced to vegetables (string beans) occurred at Darmstadt in 1904.

Geiger, Dickson, and Meyer (1922) express the opinion that

> the food and food products responsible for botulism in Germany were primarily home-preserved. It is also pointed out that the prevalence of botulism in country districts frequently is due to inadequate preservation on account of the careless and unsanitary treatment to which the raw material is subjected by the rural population. Home slaughtering and preservation of pork products in form of sausages is so universally practised in Germany that it is not at all surprising to find that about one-half of the botulism outbreaks were caused by this type of food. Inasmuch as many of the German records and histories are rather vague and indefinite, it seems, however, hardly fair to draw further comparative deductions.

In America, although meat and preserved meat products, sausages, fish, shell-fish, cheese, etc., were among those foods formerly associated with cases of botulism, the majority of outbreaks in recent years have been attributed to canned or bottled fruits and vegetables. These included apricots, asparagus, string beans, beet, olives, onions, pears, peas, spinach, and sweet corn. They were mostly home-preserved in cans or glass jars and consumed cooked or uncooked in the form of salads. 'Home-canned string beans alone accounted for 19 out of 55 outbreaks of botulism' (Topley and Wilson, 1936).

Geiger, Dickson, and Meyer (1922) record that in 33 outbreaks plant products caused 72·5 per cent of the cases, while 2·73 per cent were attributed to animal products. Commercially canned spinach and home and commercially canned string beans were responsible for one-half of the single or group family cases. The wide distribution of *Cl. botulinum* in the soils of America seems to offer sufficient explanation of the contamination of so large a variety of fruits and vegetables.

Meyer (1936) in recording the foods responsible for 261 outbreaks in the United States and Canada during the years 1899–1935, points out that

> although under-sterilised plant products were involved in 76·3 per cent of the cases, it is important to emphasise that animal products (14·9 per cent.) continue to play a rôle in the outbreaks. Among the newer foods associated in the recent fatal cases, home-canned pork, salmon and crab meat must be mentioned.

In France Bénard, Rambert, and Pestel (1943) recorded cases of botulism after the consumption of preserved goose. Scott (1943), in an abstract referring to the above, says:

> In these days when food is scarce and meats are preserved and potted at home and even provisions not quite fresh may be used, the cases of

botulism recorded here should serve a useful purpose by way of warning The food which proved to be the source of infection was goose preserve, which formed part of 'funeral baked meats', and six of those who ate it were attacked; one, a woman of 52 years, died. The symptoms were typical. Three to four hours after the meal digestive troubles began with prostration, abdominal pain, nausea and vomiting, to be succeeded some 14 hours later by ocular symptoms, paralysis of accommodation ptosis, strabismus, and a paresis of the pharyngeal muscles and difficulty of speech. Two other cases are reported in which the incubation was longer, 2–3 days, and the symptoms less severe, dryness of the throat, paralysis of accommodation and marked prostration; in a third patient the latent period was as much as six days, and the ocular symptoms the only ones observed. The cause was shown to be Cl. botulinum B, the type most common in France. The patient who died had received by way of treatment 1 c.c. of the anatoxin and 60 c.c. of antiserum on one day, and 40 c.c. more on the day following.

Jordan (1931) pointed out that

There is a significant uniformity in the type of food implicated. Fresh food, raw or cooked, is not the bearer of botulinum toxins. Practically all the reported cases of botulism have been caused by food that has been given some sort of preliminary treatment, as smoking, pickling or canning, then allowed to stand for a time, and eaten without cooking. Most of the recent outbreaks were due to home-canned vegetables processed in boiling water. Provided the food substance is not too acid or too alkaline and is shut off from free access of air, almost any food seems able to serve as a culture medium for the specific bacillus.

The foods responsible for the cases of botulism which have occurred in Great Britain were as follows.

Loch Maree, Scotland (August 1922): potted wild duck paste in glass containers (commercially preserved). The preparation of the paste was given as follows. The meat was boned and weighed, and afterwards cooked in an open cooker for an hour or two. It was then transferred to machines to cut it up and reduce it to a paste and the flavouring ingredients added. The mass was placed in large shallow pans, two feet square and three inches deep for sterilization under 10 lb. pressure for two hours at 237°–240°F. The meat was removed from the sterilizers and stirred with sterilized paddles. It was then placed in the filling machines and delivered into glasses which were capped and cooked at 210°F. for 40 minutes. Regarding the above process, Tanner (1933) remarks:

In the light of the work which has been done in the United States on the heat-resistance of the spores of Clostridium botulinum, such cooking would not destroy even some of the weaker spores, to say nothing of those which have been shown to be especially resistant to heat. The procedure for the preparation of this duck paste, together with

the chemical constitution, make it an ideal product in which Clostridium would form its toxin. If there were a few spores of the organism present in the beginning, they would be disseminated throughout the paste. The slight heating which the paste received might reduce the content of other organisms which could have an antagonistic effect on the toxin producer and thus give the Clostridium botulinum freer range.

North London (August 1935):

The incriminated 'vegetable brawn' consisted of a mixture of various vegetables (carrot, turnip, peas, beans, vegetable marrow, etc.) with ground-up nuts of various kinds, breadcrumbs, flour, herbs, spice, and hard-boiled eggs embedded in an agar jelly flavoured with marmite. Altogether some 60 jars, each of rather less than a pound, had been manufactured during the month preceding the outbreak; none of these except the two responsible for the outbreak was found to contain the specific toxin (12 examined). The vegetables were steamed for about 20 minutes before being added to the brawn mixture. The whole mixture was then placed in glass jars, sealed with airtight lids and steamed at the boiling-point of water for 2 hours, the same process having been used for 30 years. Cl. botulinum was isolated from the remains of the nut-meat brawn consumed by the patients who died of botulism. Its characteristics were those of Type A [Ann. Rep. C. Med. Officer Min. Hlth., 1935, p. 151].

North London (August 1935): steak pie. *Cl. botulinum*, Type B, isolated from the pie. This was the first occasion this type has been obtained in this country in connection with a human case.

London (1947), five suspected cases of botulism and one death: the food consumed by all the cases was macaroni and cheese with sauce. No *Cl. botulinum* was isolated. Battersea, London (1955): pickled fish and vegetables from Mauritius, consumed by two students. *Cl. botulinum*, Type A, isolated.

Physical Appearance (Signs of Spoilage)

The early history of botulism records that the contaminated foods were spoiled or decomposed. In recent years, although this condition has not been observed in every instance, in the majority of outbreaks the foods showed more or less marked changes from the normal or were noticeably spoiled. In the case of home-preserved fruits and vegetables in jars, bubbles of gas were present and the liquid squirted out when the tops were unscrewed. The contents had a distintegrated appearance, a bitter taste, and gave off a smell like rancid cheese or butter. When the food was preserved in cans, these were sometimes blown and the contents had a mushy appearance and rancid odour. In occasional instances, however, the canned food presented no abnormality in consistence

or odour although the toxin was present. In other cases, the rancid smell was only noticeable when the food was heated, while the physical disintegration was slight. Cloudiness of the brine or liquor may be the only sign of bacterial activity.

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Chapter XXII

ILLUSTRATIVE OUTBREAKS

The Loch Maree Tragedy in Scotland

The first recorded outbreak of botulism in the British Isles, as before mentioned, occurred at Loch Maree, Gairloch, Ross-shire, in August 1922. A party of visitors staying at a local hotel went out fishing on the morning of 14 August and during the day partook of some sandwiches which contained potted wild duck paste.

About 3 a.m. on the 15th one of the visitors was taken ill, and later several of the others complained of illness. The first death occurred at 9 p.m. on the 15th, and all 8 persons died during the ensuing week.

The following tables from Leighton's work on botulism give a summary of the symptoms of each case, together with period of onset and duration of illness.

TABLE OF SYMPTOMS

Patient	*Age*	*Dizziness*	*Double Vision (Diplopia)*	*Paralysis of Eyelids (Ptosis)*	*Paralysis of Speech*	*Paralysis of Swallowing*	*Respiratory Distress*	*Reflexes Diminished*	*Cardiac Failure*	*Intense Restlessness*	*Vomiting*	*Pupils Dilated*	*Headache*	*Some Pain*	*Diarrhoea*	*Fever*	*Loss of Consciousness*
Mr. S.	70	×	×	×	×	×	×	×	×	×	×	—	×	—	—	—	—
Mr. W.	66	×	×	×	×	×	×	×	×	×	×	—	—	—	—	—	—
Mrs. D.	56	×	×	×	×	×	×	×	×	—	—	—	—	—	—	—	—
Mr. D.	60	×	×	×	×	×	×	×	×	×	—	—	—	—	—	—	—
Mr. T.	22	×	×	×	×	×	×	×	×	—	×	×	—	—	—	—	—
Mrs. A.	45	×	×	×	×	×	×	×	×	×	×	—	×	×	—	—	—
K. McL.	35	×	×	×	×	×	×	×	×	×	—	×	—	×	×	—	—
J. McK.	40	×	×	×	×	×	×	×	×	—	—	—	—	—	—	—	—

It will be seen that the shortest interval between the ingestion of the incriminating food and the onset of the symptoms was about 14 to 18 hours and the longest 44 hours.

Samples of the remains of some of the wild duck paste, as well as that in one of the sandwiches, were bacteriologically examined

at the University of Bristol by Bruce White. A long series of cultures were instituted on various media and the organisms grown under both aerobic and anaerobic conditions.

PERIOD OF ONSET AND DURATION OF ILLNESS

(Lunch assumed at 1 p.m., 14 August)

Patient	*Age*	*Onset, Before Symptoms Appeared*	*Duration, After Symptoms Appeared*
Mr. S.	70	15 hours	17 hours
Mr. W.	66	14 ,,	$21\frac{1}{2}$,,
Mrs. D.	56	17 ,,	18 ,,
Mr. D.	60	17 ,,	6 days
Mr. T.	22	20 ,,	$24\frac{1}{2}$ hours
Mrs. A.	45	18 ,,	46 ,,
K. McL.	35	26 ,,	46 ,,
J. McK.	40	44 ,,	$5\frac{1}{2}$ days

According to Bruce White's report:

Of these cultures, all the sandwich meat cultures and all the wild duck cultures were found to be terribly pathogenic to mice when minute quantities were injected subcutaneously. The cultures giving positive results were now closely scrutinised. Microscopically the wild duck cultures had every appearance of purity, consisting entirely of large bacilli producing egg-shaped terminal spores. An anaerobic sporing bacillus had been isolated from the wild duck paste and was highly pathogenic to mice. As soon as full cultures had been set up experiments were initiated to test the toxicity or otherwise of the samples themselves.

The conclusions which the bacteriologist arrived at were as follows:

1. The wild duck paste contained a potent toxin, the action of which is inhibited by botulinus (Type A) anti-toxin.
2. The anaerobic spore-bearing bacillus isolated from the 'wild duck' paste produces a similar toxin which is likewise counteracted by botulinus (Type A) anti-toxin.
3. The identity of the wild duck bacillus with B. botulinus (Type A) seems established, as also the identity of botulinus (Type A) toxin and that of the wild duck paste.
4. It seems certain that the wild duck paste and the sandwich were the only toxic foodstuffs submitted for examination.

Further interesting experiments were subsequently carried out upon mice. A very minute quantity of the wild duck paste was made into an emulsion and injected into three mice. All the mice died. Similar injections were made into three other mice, but in

addition each was given a dose of anti-toxin along with the poisonous emulsion. These mice remained completely protected by the anti-toxic serum.

The bacteriological examinations and the experiments carried out definitely proved the presence of *B. botulinus* and its toxin in the suspected wild duck paste, and that the outbreak was due to the ingestion of the sandwiches containing this paste.

Home-Preserved Asparagus in Seattle

On Saturday, 24 November 1917, Mrs. E. of Seattle, Washington, opened a jar of home-preserved asparagus and cooked half of the contents (Dickson, 1918). None of the persons who ate the cooked asparagus suffered any ill-effects. On the following evening she 'warmed up' the remainder of the asparagus from the jar by placing it for a few minutes in warm but not boiling water. Her husband stated that this asparagus did not taste very good, but he ate it all. On Tuesday afternoon Mr. E. complained of disturbance of vision, was nauseated and vomited. On Wednesday morning he was very weak, vomited again after taking food, and had severe diarrhoea. The diarrhoea continued during the day and following night, and during the night the patient complained of cramps in the legs. There was no abdominal pain during this time and no disturbance of sensation. During Wednesday afternoon he began to have difficulty in talking. On Thursday Mr. E. was unable to sit up because of weakness, and he complained that he could not hold up his head. He was unable to speak intelligibly and he complained of dryness in the mouth and pharynx. During the afternoon he began to have difficulty in swallowing and by evening 'all the water returned through his nose'. He had much difficulty in clearing thick, tenacious mucus from the pharynx and had severe strangling spells when he attempted to swallow. There was no disturbance of mentality, no pain except the cramps in the legs, and no fever. He was found dead in bed early Friday morning, about 2 hours after he had succeeded in swallowing a small quantity of milk.

On Thursday, 29 November, another jar of the same lot of asparagus had been opened and served cold as salad at the Thanksgiving dinner. Two persons partook of the salad and both developed symptoms and died. The remnants of the salad was fed to the chickens, and all the chickens developed typical symptoms of limberneck and died. Bacteriological examination was made of

the contents of the crops and gizzards of 10 of the chickens and from 6 of them a virulent strain of *B. botulinus* was isolated.

The asparagus had been purchased in the open market and canned at home by the method described in the pamphlet issued by the manufacturer of the glass jars which were used, with the exception that it was not parboiled or blanched before it was packed into the jars. The asparagus was washed in cold water, packed into 1-pint and 1-quart jars, which had been boiled, and covered with cold water to completely fill the jars. One half teaspoonful of salt was added to each jar and the covers loosely applied. The jars were immersed to the neck in a wash-boiler, which had a tightly fitted cover, and were allowed to remain for 3 hours after the water began to boil actively. On removal from the boiler, the jars were tightly sealed and placed in a dark closet.

It is interesting to note that in the above outbreak none of the persons who partook of the cooked asparagus suffered any ill-effects, whereas the man who ate the remainder of the asparagus from the jar uncooked developed the typical symptoms of botulism.

Home-Preserved Apricots in California

On Sunday, 27 January 1918, a party consisting of 9 persons, 5 adults and 4 children, had supper together near Madera, California (Dickson, 1918). The supper consisted of fresh pork, brown beans, bread, butter, milk, and home-preserved apricots. It was noted that the apricots had a peculiar taste, but 8 of the party ate some of them. The only member of the party who escaped illness was the one who did not eat any of the apricots.

On Tuesday morning, 29 January, 3 of the children, aged 3½, 5 and 14 years, respectively, complained of seeing double, and 3 of the adults complained of dizziness. The 3 adults also developed diplopia during the day. The fourth adult first showed symptoms of illness on Tuesday night, and the smallest child, aged 13 months, became ill on Wednesday evening. One of the children died on Wednesday morning, 30 January, two on Wednesday evening and and one on Friday morning. Two of the adults died during the night of Thursday and the remaining two apparently recovered after a prolonged illness.

The symptoms of all the patients were practically identical except in degree of severity. In all there were dizziness, weakness and inco-ordination of muscular movement, early disturbance of vision with blepharoptosis, mydriasis and diplopia. difficulty in swallowing and talking, and strangling spells induced by attempts

to raise thick mucus from the pharynx or to swallow. In one case, one of the patients who recovered, there were initial diarrhoea and vomiting which occurred from 12 to 15 hours before the onset of the eye symptoms, but in none of the other cases were there acute gastro-intestinal manifestations. In all the cases there was persistent constipation.

On Wednesday, 29 April, the portion of the apricots which remained in the jar was thrown to the chickens. On Thursday several chickens showed signs of limberneck and some of them died, and by Friday afternoon over 25 chickens and 1 turkey had died. A wild canary with similar symptoms was found lying under a tree and it died a few hours later. Bacteriological examination of the contents of the gizzard of one of the chickens revealed the presence of a strain of *B. botulinus* which produces a virulent toxin when grown in suitable culture mediums.

Home-Pickled Herring in Canada

Dolman, Chang, Keer, and Shearer (1950) mention 2 cases (1 fatal) of botulism caused by the consumption of home-pickled herring, caught near Vancouver, B.C. *Cl. botulinum*, Type E, was isolated from the stomach and jejunal contents of the fatal case and from a fragment of discarded herring-bone. The observers point out that

> this episode may illustrate a serious risk inherent in pickled herring (and other forms of uncooked fish) which is insufficiently appreciated by persons of varied heritage, e.g. Jewish, Scandinavian, who enjoy such food. Even in the most careful hands, fish are exposed to numerous opportunities for bacterial contamination. Their reputation for being seldom implicated in any kind of bacterial food poisoning is perhaps due more to the rapidity with which fish undergo spoilage than to the effectiveness of any special hygienic precautions.

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Chapter XXIII

PREVENTION AND CONTROL OF BOTULISM

THE problem concerning the preventive measures against botulism—which stands alone as a type of food poisoning—does not appear on the surface to be a very difficult one at the present time, as, according to statistics, since the Loch Maree outbreak in 1922, up to and including 1955, there have been only 5 deaths recorded definitely due to botulism in Great Britain. There are, however, certain important general precautions and measures of control which, from time to time, have been evolved as a result of intensive experimental work.

Botulism is endemic in other parts of the world where large quantities of canned and preserved foodstuffs are produced, both for home consumption and for export, but more especially in those countries where much home canning and preserving are carried on.

In England, Scotland, and Wales, owing to the high standard of efficiency maintained in the inspection of all consignments of canned food at the ports of entry, the community enjoys protection against the possibility of the disease from these sources.

In the United States botulism formerly offered a serious menace to the canning industry, but through extensive research and experimentation over a long period, to ascertain the conditions of heating necessary for different foodstuffs to render them safe for consumption, methods were evolved to eliminate the disease. In commercial canneries, pressure cookers employing live steam are in use, and by scientific tests a correct processing time is determined for each important foodstuff. As a result of the use of this special apparatus and by the enforcement of various sanitary precautions the incidence of botulism from commercially canned foods has been reduced to a minimum.

Meyer (1931) said that observations of the preceding ten years left no doubt that all canning methods, whether commercial or home, should aim at absolute sterility of the product to ensure freedom from *Cl. botulinum* or *Cl. parabotulinum*. 'In case this pre-requisite cannot be met, acidification with citric acid, or with a mixture of acetic and citric acids, to a *p*H of at least 4·5 with subsequent heating of the product at 100° for a short period, should be practised. Such vegetables as artichokes, chillies,

mushroom sauces, etc., can now be well preserved by the procedure of acidification.'

Mention of a few recent publications, issued in the United States on this important subject, may prove useful for reference by firms in Great Britain engaged in, or contemplating, the preservation by heat of foodstuffs in airtight containers:

Processes for Non-acid Canned Foods in Metal Containers. Bulletin No. 26L (8th edn.), Nat. Canners' Assoc. Research Lab., Washington, D.C., 1955.

Mathematical Solution of Problems on Thermal Processing of Canned Food, by Charles Olin Ball, Research Div. American Can Co., Maywood, Illinois, 1928.

Thermal Processes for Canned Marine Products, by O. W. Lang, Univ. of California Publications in Public Health, 1935.

Sterilisation in Food Technology by C. O. Ball and F. C. W. Olson, publishers McGraw-Hill.

Processes for Low-Acid Canned Foods in Metal Containers, Bulletin 26-L (6th edn.), Natl. Canners Assn., Research Lab., Washington, D.C., 1946.

Processes for Low-acid Canned Foods in Glass Containers, Bulletin 30L (2nd edn.), Nat. Canners' Ass., Research Lab., Washington, D.C., 1955.

Laboratory Manual for the Canning Industry prepared and published by the Nat. Canners Ass., 1956.

Home Canning of Fruits and Vegetables, Home and Garden Bull. No. 8, 1947, United States Dept. of Agriculture, Washington, D.C.

Principles and Methods in the Canning of Fishery Products, Research Report No. 7 by Norman D. Jarvis, U.S.A. Dept. of the Interior, Fish and Wildlife Service, 1943.

Community Canning Centers, Misc. Publication No. 544, Production and Marketing Admn., U.S. Dept. of Agriculture, 1946.

Home Canning of Meat, Home and Garden Bull. No. 6, 1951, U.S. Dept. of Agriculture, Washington, D.C.

The Chief Medical Officer of the Ministry of Health in his Annual Report (1935) pointed out:

It is important that firms engaged in this industry should be well-informed on this subject and should be equipped with appliances which are capable of doing the necessary sterilisation and are properly operated to this end. Some firms may not devote the attention which they should to this aspect of their business, and if this is so they constitute a menace to the consumer and to the reputation of the trade as a whole.

Home-Canning and Preservation

The fact that *Cl. botulinum* occurs naturally in the soil and is widely distributed throughout the world, makes any attempt to avoid initial contamination of fruits and vegetables difficult. The organism thrives and multiplies on decaying vegetation, and whenever spoilage of the raw product occurs, any spores present may rapidly increase in numbers.

The home canner naturally purchases vegetables or fruits in the cheapest market, and these may have been stored for several days. Poor quality especially of vegetables that are heavily contaminated, constitutes the principal source of trouble.

It may be of interest in passing to quote Leighton's (1923) comments on the subject:

> It is well known that a large number of ordinary foodstuffs are quite suitable material for the growth of the bacillus in them, and for the formation of its toxin, provided that the special conditions necessary are added. It so happens that these conditions are found nowhere better than in the airtight container of these preserved foods. If the organism itself is sealed up along with such food without being killed, or if the spores of the organism are so sealed up, having escaped killing on account of an insufficient temperature being applied to the container in the process of sterilisation, then we have all the conditions required for the production of a dangerously toxic product. In such a case the air has been driven out of the container, and with it the free oxygen. The nutrient medium necessary for the growth of the organism is found in the foodstuff itself, and it is only a question of time for the production of the toxin. If the bacillus under these circumstances starts growth there may be production of gas within the container, and other changes which on the container being subsequently opened may be obvious to an observer, and which ought to cause the immediate rejection of the food as spoiled. But it cannot be said that this is always the case, for a number of observers state that in certain cases where the organism has produced its toxin, the preserved food on being opened has shown no obvious change either in smell or appearance.

Generally speaking, in home-canning and preserving the temperature attained in heating is, as a rule, too low to kill any spores of *Cl. botulinum.*

Fractional sterilization, which is sometimes practised, is unreliable, because the spores may not develop in the meantime (Burke, 1919). It is only with steam under pressure that the spores of the bacillus can be destroyed.

A fair amount of home-canning and bottling of fruits and vegetables is carried out in this country, and much useful information and instruction are afforded to individuals attending lectures

and demonstrations given by educational bodies. Moreover, useful publications on the subject have been issued from time to time by the Ministry of Agriculture, Fisheries, and Food. *Bulletin* No. 21 (8th edn., 1954) deals with the domestic preservation of fruit and vegetables. They also publish *The A.B.C. of Preserving* (3rd edn., 1955) and Advisory Leaflet No. 434, *The Home Freezing of Fruit and Vegetables* (1955). In addition the Fruit and Vegetable Canning and Quick Freezing Association, Chipping, Campden, issue a Technical Bulletin (No. 4, 1956), *The Processing of Canned Fruit and Vegetables.*

While the growth of *Cl. botulinum* and the formation of its toxins are inhibited by acid foods, such as fruits and tomatoes (these can be processed at or near the temperature of boiling water), it is essential for the satisfactory sterilization of non-acid foods, such as peas, beans, and practically all vegetables, that more elaborate equipment should be employed to obtain the high temperatures such as are produced in steam-pressure cookers. These destroy the spores of the organism and prevent any subsequent bacterial growth.

Meyer (1934) remarks:

> If there is no pressure cooker available in home-preserving of non-acid foods, it is safer to substitute dehydration, salting or pickling for canning.

It has been said that dissolved tin, such as results from the use of unlacquered food cans, inhibits the growth (Scott and Stewart 1944).

Hall (1943) says:

> It is recognised that while the pressure-cooker, properly operated provides the easiest and best method of home canning, there is likely to be a shortage of such cookers. Correct operation should be emphasised we have recorded three outbreaks of botulism caused by food supposed to have been sterilised in pressure-cookers.
>
> Emphasis should undoubtedly be placed upon the following items
>
> 1. Processes:
>   - (*a*) Careful selection of sound produce.
>   - (*b*) Careful cleansing, and when indicated, blanching of produce as well as general cleanliness to minimise the bacterial load to be sterilised.
>   - (*c*) Principles and correct application of intermittent sterilisation as a valuable and available substitute for cooking under steam-pressure.
>   - (*d*) Use of other methods of preserving food, notably drying salting, and pickling in which there is little or no danger from botulism.

2. Consumption of home-canned foods:
   (a) Significance of turbidity, gas-production, softening and odour as criteria of spoilage.
   (b) Danger of eating, or even tasting, freshly opened home-canned foods, especially if signs of spoilage are present.
   (c) Fact that certain foods, notably beets, chili, sometimes beans, and possibly other foods, may show no easily recognisable signs of spoilage even though botulinus toxin is present.
   (d) Destruction of botulinus toxin by always boiling home-canned foods for at least 5 minutes before serving.
   (e) Harmlessness of the spores of Bacillus botulinus.
3. Safe methods of disposal of contaminated foods by boiling in strong lye-water to avoid:
   (a) Killing poultry and other domestic animals.
   (b) Excessive pollution of the soil with the spores of Bacillus botulinus.
   (c) Loss of usable containers.
4. In case an outbreak occurs:
   (a) Character of symptoms of botulism and other forms of food poisoning.
   (b) Prompt reporting of suspicious symptoms to physicians.
   (c) Diagnostic value of symptoms in fowls and other domestic animals in case humans have tasted same food.
   (d) Inadequacy of botulinus antitoxin for treatment of advanced botulism.
   (e) Saving of remnants of food for epidemiological and laboratory studies of food poisoning.

Canning and Preserving Essentials

Here are a few important points and precautionary suggestions in connection with home-canning and preserving.

Vegetables and fruits must be fresh and sound. The former should be young and washed free from dirt and grit and preserved as soon as possible after gathering. If the raw foodstuffs are not required for immediate use, they must be stored under conditions that will prevent deterioration.

Acid-pickled foods require at least 2 per cent of acetic or citric acid (with a pH of 4·0). Brine foods should contain not less than 10 per cent of common salt.

Ingram and Robins (1951), in a discussion on botulism in relation to acid foods, concluded from their survey of the evidence that it does not warrant unreserved acceptance of the view that the growth of *Cl. botulinum* can occur below pH 4·5 whether accompanied by other microbes or not. A pH of 4·5 does in fact afford very good protection against *Cl. botulinum* intoxication.

Vegetables preserved by the 'cold pack' method must never be served as salads unless they have been previously boiled.

These simple and inexpensive methods of preservation have one safeguard, i.e. the food is not ready to be served from the container but requires soaking in water and sometimes subsequent boiling, which is of course the most effective precaution.

All bottles and cans of preserved food must be carefully examined before opened. There should be no bulging of the tops of the bottles and no escape of gas or liquid when opened. The ends of the cans should be flat or curved slightly inwards; the inside, smooth and not corroded and the odour characteristic of the product. Never taste food to discover spoilage.

Preserved food which by a rancid or butyric-like odour arouses suspicion or shows the least evidence of deterioration must be excluded from consumption.

Jars, bottles, or cans suspected of being unsound must never be disposed of indiscriminately, as they may contaminate the soil or even cause botulism in animals or poultry. Burn or bury all spoiled food. In handling suspected foodstuffs care should be taken to prevent it coming into contact with cuts and abrasions on the hands, as according to Geiger (1924) the toxin may be absorbed by broken skin areas, mucous surfaces, and fresh wounds.

Regarding spoilage, Dickson (1918) states:

> It is a point of considerable importance that foodstuffs which are contaminated with the toxin of bacillus botulinus may not appear sufficiently spoiled to ensure their being discarded. The vegetables usually have an unpleasant odour and may show bubbles of gas on the surface, but they are not apt to be discoloured or soft and may even appear to be especially well preserved. It should be thoroughly understood that an extremely virulent toxin may produce but little change in the appearance of the food, and the common practice of tasting canned stuff to see whether it is fit for use should be discouraged. All canned food should be discarded if there is any indication that it is even slightly spoiled (this is even more important with home-canned food), and under no circumstances should it be eaten or even tasted before it has been cooked.

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PART V

Chapter XXIV

CANNED FOODS AND THEIR INSPECTION

by Cecil Ash, F.A.P.H.I., M.R.S.H.

Historical

Napoleon may have observed that an army marches on its stomach, but it was Nicholas Appert (1752–1840) who helped to keep full the military stomach by the first successful large-scale methods of food preservation from which the modern canning industry is descended. Appert concluded that air was responsible for putrefaction and that if heat was applied to foods in sealed jars they would have an indefinite life. He packed meats, fruits, and vegetables into wide-mouthed jars, sealed the tops with corks and wire and sterilized in a *bain-marie*. In 1806 foods so treated were supplied to the French Navy. The success of his method was acknowledged in 1809 by an award of 12,000 francs presented by the Bureau Consultatif des Arts et Manufactures. In 1810 Appert published his famous work under the title *L'Art de conserver pendant plusiers années toutes les substances animales et végétales.*

John Hall and Bryan Donkin, in England, developed Appert's methods of preservation at Hall's foundry in Dartford, Kent, applying the patents of Augustus de Heine and Peter Durand by which iron containers were substituted for glass. Donkin (1768–1855) is considered the father of the modern canning industry. From their factory in Bermondsey the firm of Donkin, Hall, and Gamble were soon supplying the Army and Navy with canned foods.

In 1841 a method was evolved for testing the keeping properties of these commodities in the tropics. Sealed cans were placed for one month in a chamber at a temperature of 90°–110°F., 'blown' cans being rejected.

The manufacturers exhibited canned foods at the Great Exhibition in 1851 under the name of Messrs. John Gamble. A description of the exhibit gives an idea of the canning methods employed:

> The process consists in placing the partially cooked provisions into tin canisters, with a little bouillon or juice of the meat, then soldering

on the covers, which have a small hole perforated therein. The tins are after this immersed to a great portion of their depth in a saline bath heated above the boiling point of water and left therein until the air has been expelled as completely as possible by the steam generated within them; the hole in the cover is now hermetically closed with a little solder, the tin being momentarily touched with a damp sponge to stop the egress of steam. The minute portion of oxygen still remaining in the tins enters into combination with the animal or vegetable matter at the induced temperature and thus further change is prevented. After sealing of the tins, they are submitted to the ordeal of the testing room, heated to a temperature above 100°F.; if putrefaction takes place, the generated gases burst the tins, but those which pass uninjured remain perfectly good. [*Historic Tinned Foods*, 1939, International Tin Research].

It was doubtless the inability to recognize the part played by heat in the destruction of bacteria which caused the many failures experienced in the early days of the industry, notably those of Goldner. This led to the appointment of a Royal Commission to enquire into the wholesale condemnations of meat canned for the Navy. The correlation between the size of pack and liability to putrefy was noted, though not then understood. Although the industry spread to New South Wales and the United States, it was not until 1895 that bacteriology was directly applied to the problems of food canning when Prescott and Underwood in America carried out investigations upon the spoilage of canned corn. Hitherto Pasteur's work on fermentation had not been correlated to the practices of canning.

Just as the Napoleonic Wars had initiated the industry, so the American Civil War stimulated it and the economic expansion of the later nineteenth century (especially mechanical production of cans and the extension of railroad systems) established canning as one of the most important enterprises of the United States. Once the scientific principles underlying successful canning technique were understood, satisfactory processes were established and temperatures for commercial canning of all classes of food were standardized.

Introductory

Canning is a method for preserving carefully selected and prepared foodstuffs, dependent for its success on the expulsion of air from and sealing of the can, the destruction by heat (at definite temperature for a prescribed time) of spoilage organisms, efficient cooling, satisfactory storage and distribution. This basic objective is reinforced by measures to retain as far as possible the

natural appearance and properties of the food, so that the product marketed is reliable, safe, and wholesome. The canning industry to-day has generally achieved all these objectives and in consequence cases of bacterial food poisoning are infinitesimal compared with the very large quantities of canned foods consumed in Great Britain.

Savage (1939) pointed out that

> canned foods share with all other foods the risk of being a vehicle to cause food poisoning. From a study of all the data, it can be definitely stated that canned foods are now considerably less liable than ordinary foods to be a source of food poisoning. This is conspicuously so for the more dangerous outbreaks associated with the presence of living bacilli. Liability to cause the milder outbreaks of toxin type still exist but is being reduced, and even for this type their incidence, bulk for bulk, is much less than that of other foods.

Between 1950 and 1955 recorded cases in England and Wales of outbreaks of food poisoning in which the vehicle was canned (including bottled) foods fell from 59 to 15. Canned meat and canned fish were the vehicles chiefly implicated, though occasionally canned fruit and vegetables appeared in the list. There is evidence that in some cases the causative organism was present in the can. As the Report of the Public Health Laboratory Service ('Food Poisoning in England and Wales', 1954) comments,

> it is not suggested that the risk of food poisoning from this source is of great importance in relation to the number of cans [of meat] eaten, but canners should study means to close this occasional loophole in the safety of canned foods.

While this record of comparative safety is to the credit of the canning industry we should not ignore the work of the port health authorities in the inspection of incoming foods, resulting at times in the condemnation and destruction of large quantities of canned foods. In some instances, however, the importer undertakes to re-export an unsatisfactory consignment, guaranteeing that none of the foods will be re-imported into this country. At the Port of London alone, canned foodstuffs destroyed in 1954 amounted to 174 tons, in 1955 to 59 tons, and in 1956 to 112 tons. Supervision of processes in canneries in this country falls within the duties of local health authorities, who are entitled to share, with the industry, the credit for this excellent record.

Scientific Principles of Processing

Whatever foodstuff is canned the basic principles and processes remain substantially the same. There are four main stages in the operations:

(1) Preliminary preparation prior to filling.
(2) 'Exhausting' to remove as much air as possible from the filled can.
(3) 'Sterilization' of sealed cans.
(4) Cooling.

The object is the destruction of organisms liable to cause spoilage, packaging so as to deny further access, and maintenance of a bacteria-free environment until the contents of the cans are consumed. It should therefore be noted that 'sterile' and 'sterilization' in industry do not necessarily mean total destruction of all organisms and spores. Efficient processing will destroy spoilage organisms and pathogens; such a process achieves 'commercial sterility' and the words 'sterile' and 'sterility' will have that meaning in this chapter.

Preliminary Preparation

Apart from reducing to convenient size, sorting, and grading, preliminary treatment may include operations such as peeling (fruit and vegetables), stoning (fruits), brining (some meat and fish), and most foods will be washed and trimmed. Peeling may be manual, chemical, or mechanical. Stoning, sorting, grading, trimming, and washing may be performed manually or mechanically. Dissection and brining involve manipulation. Whatever the initial bacterial load of raw material entering the plant, opportunities for distributing the original flora or adding additional contaminants abound during the treatment prior to actual canning. Enlightened operators take the view, however, that they do not wish their products to be cemeteries for bacteria, and the less enlightened find that failure to exercise reasonable bacteriological control during preliminary processes is likely to render more difficult the 'sterilization' itself. The greater the initial contamination in a given food the longer the period of heating required to sterilize it (Cameron and Yesair, 1931).

It has been shown that spores introduced in their natural environment are more resistant than those developed on artificial media. Time and temperature calculations made in the laboratory using cultured organisms of the type expected to be encountered may prove unduly optimistic. The classic investigation of this type of problem was that of Cameron, Esty, and Williams (1936) into spoilage of canned meat. Conversely, a high standard of hygiene and bacteriological control during preliminary treatment may permit some reduction in the theoretically calculated time-

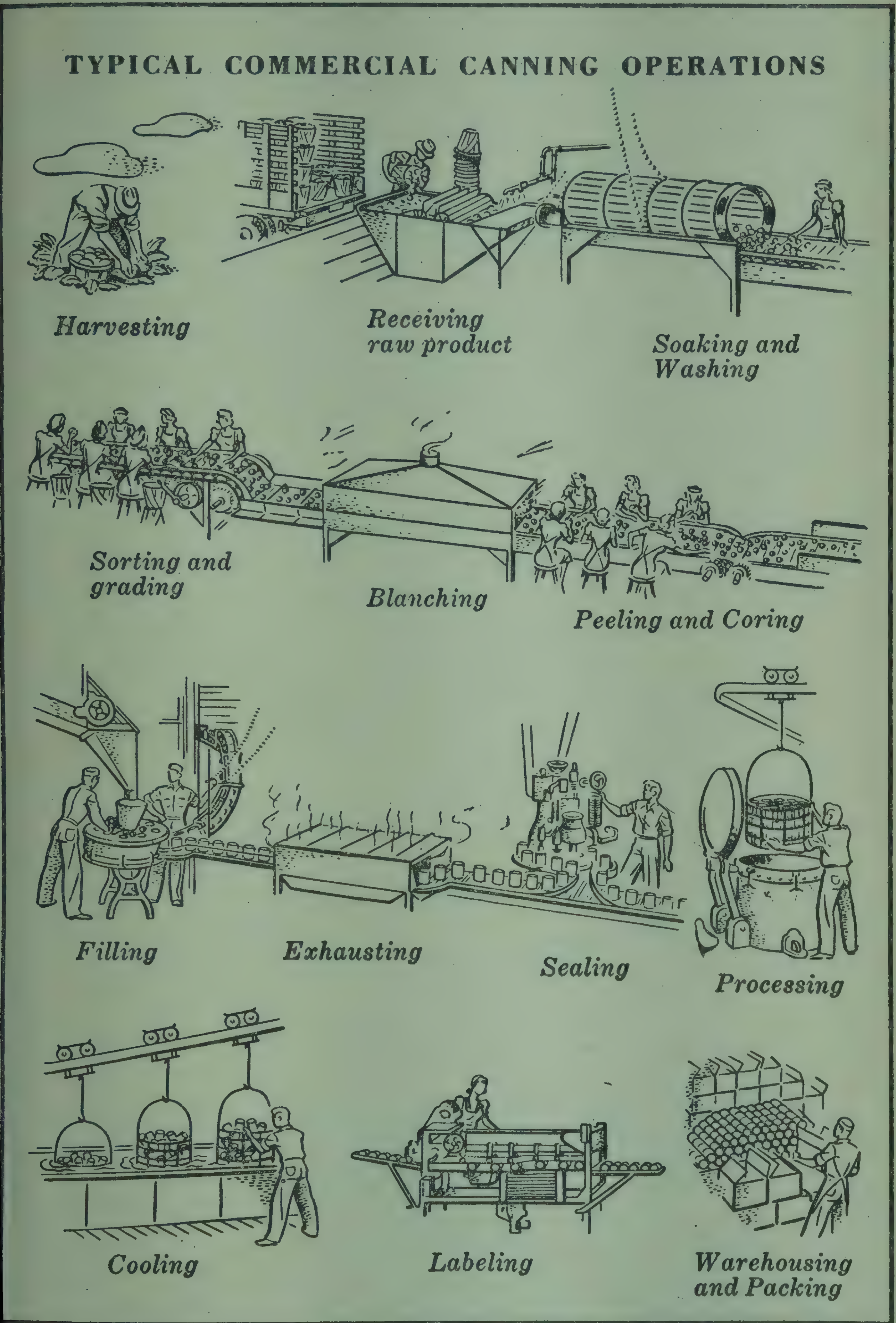

FIG. 14. TYPICAL COMMERCIAL CANNING OPERATIONS (courtesy *American Can. Co.*).

temperature curve, a factor of some importance where the marketability of a pack may be adversely affected by high temperature or prolonged exposure.

Essential preliminary treatment may have some bactericidal effect—for example, the blanching of fruit, vegetables, and meat to inactivate enzymes will reduce to some extent the bacterial load, though thermophiles will not be affected. Salt, sodium nitrate, and vinegar which may be introduced as part of the preparation will have an inhibitory effect, if used in appropriate concentration. Even such a laudable operation as washing raw material is of bacteriological significance for by providing 'free' (i.e. available) moisture it may facilitate proliferation of organisms especially if there is an appreciable time lag at significant temperatures between preparation and sterilization.

If canners appreciate that during pre-process treatment the same sanitary and hygienic control is necessary as for open-pack products, the finished article will not suffer in marketability or safety. Where they are acquiring raw material from sources beyond their control they may be at some risk from the bacteriological condition of the items reaching them, such as carcass meat, although by relating payment to bacteriological quality, as in the dairying industry, this hazard might be reduced. In some countries the common practice is to associate the cannery with the abattoir, thus extending bacteriological control in either direction; in this country such developments are as yet comparatively rare.

Measures entirely within the manufacturer's competence include the sanitary construction of premises and equipment, personal hygiene of employees, the adoption of standard hygienic techniques and in particular the reduction to a minimum of manual handling.

Exhausting

Baumgartner (1943) lists the objectives of exhausting as (*a*) reduction of strain on the can through expansion of air during heating, (*b*) reduction of internal corrosion through removal of oxygen, (*c*) creation of vacuum when can is cooled.

Heat-exhausting is practised by heating the filled can immediately before sealing, displacing air in the head-space by water vapour. Expansion of contents on heating and contraction on cooling have a bearing on the success of the operation, so that head-space as well as temperature are important; too much will result in inefficient exhausting, too little will produce 'overfills'

and strains during sterilization. Baumgartner recommends heat-exhausting for canned meat pastes and similar products in which air is occluded and for unbleached vegetables and fruits, the cells of which contain respiratory gases. Mechanical exhausting, suitable where these problems do not arise, is carried out by applying a high vacuum to the apparatus in which the can is sealed, after filling cold. Exhausting has a bacteriological significance in that the reduction of free oxygen will inhibit the growth of aerobic organisms, though anaerobic thermophiles will survive in vacuumized foods and some spore-forming thermophiles which may cause spoilage can develop despite the comparative lack of oxygen, if temperature and conditions are favourable.

Processing

After exhausting and closing, cans are heated by saturated steam or steam-heated water in retorts, under strict time and temperature control. Best results are obtained where the whole of the air is replaced by saturated steam, which readily transfers its heat of vaporization to the cans on which it condenses.

Strict time and temperature control are necessary for efficient processing and control of retorting should rely on the agreement of temperature and pressure readings, preferably permanently recorded.

Cooling

Modern technique is to replace steam in the retort by compressed air and then to flood with water while the air pressure is maintained; otherwise retort pressure must be slowly reduced to that of the atmosphere prior to spraying by or immersion in water. Cooling is necessary to obviate overcooking but these safeguards prevent distortion of cans, which must retain sufficient heat to dry rapidly after cooling, otherwise the containers will corrode. Cooling water should be of potable quality to guard against recontamination via strained seams.

Spoilage of Canned Foods

Even when processing is efficient, spoilage may occur from time to time. Some foods, such as hams canned whole, are of a nature in which even 'commercial sterility' cannot be attained. Here a reduction of the bacteria is achieved by heat, and temperature control of subsequent environment is necessary to inhibit

growth of the remaining organisms. Such cans should be endorsed 'store only under refrigeration'. Hobbs and Ingram (1954) recommend a temperature of 5°C. (41°F.).

Spoilage from bacterial activity will arise either from survival of initial flora or from contaminants during preparation or after processing (resultant on strained seams). Even with extreme care, commercial sterilization will permit the survival of thermophiles or their spores, chiefly 'flat sours'. Jansen and Aschehoug (1951) in their investigations concerning spoilage in canned foods, found that flat-sour producing organisms were identified with *B. coagulans*, *B. circulans*, *B. circulans-B. alvei* intermediates. The acid and gas producing organisms were identified with *B. subtilis*, *B. subtilis-B. pumilus* intermediates, and *B. macerans*. Temperature of growth is between 100° and 130°F. These thermophilic aerobes (or flat-sour bacteria) produce acid souring of the contents of the can without gas production. Thermophilic anaerobes, however, produce acid and gas and blown cans. The anaerobic sulphide spoilage organisms produce H_2S gas which dissolves in the moisture present in the can and blackens the contents, which emit the characteristic odour of rotten eggs, but like flat-sour bacteria do not cause any external physical indication. Thermal death times of up to 50 minutes at 245°F. have been recorded of spores of some thermophilic strains (Bashford, 1942).

The evidence associating salmonella and staphylococcal infections and toxins with conditions existing prior to opening of the cans is imperfect, although there is a possibility of access via strained seams. Whatever the mode of access of these organisms or toxins, physical change in the food is rarely, if ever, recognizable.

Wildman, Nicol, and Tee (1951) recorded an interesting outbreak of food poisoning involving 59 persons who were infected by the consumption of imported canned cooked ham.

Inspection

Although, theoretically, the contents of a properly processed can should remain indefinitely sound, subject to satisfactory control of environment (notably temperature and humidity) during storage, ideal conditions of distribution are not always maintained and thermophilic survivors can produce changes in contents.

It is conventional to assume a safe store life, in this country, of 5 years for canned meats, galantines, sausages, fish and soup (except tomato soup); 2 years for vegetables and tomato soup; 1

year for fruits (though peaches may remain sound for 2–3 years) depending on efficiency of processing (lacquering is the critical feature with fruits such as cherries); 6–9 months for sweetened condensed milk (unsweetened, 3 years). Fruits do not normally become unfit at the expiry of these time limits, but delicacy of texture and flavour is lost; with sweetened milk the tendency is for 'candying' to take place due to crystallization of sugar.

Judgment on consignments of canned foods at port of entry are based on the results of examination of a percentage of the cases in the total consignment—usually 10 per cent. According to the particular port of entry, the rejection of a percentage of the sample (varying usually between 1½ and 2½ per cent of the 10 per cent) requires detailed examination of the whole consignment. The reason for this variation is obscure and a uniform standard at all ports would be equally advantageous to the public and the importers. Inland, the need for examination of complete consignments is less likely to arise, but where evidence arose of an appreciable amount of unsoundness in a particular consignment it would be usual to follow a similar procedure. Where canned food is stored in considerable amounts on service account, very much shorter store life is accorded to the various items by the responsible victualling departments. On expiry of the 'service store life' it is usual to refer surviving items to the civilian authority for physical examination, items which satisfy being disposed of as surplus, usually reaching the public via 'cut-price' merchants.

Methods of Examination

The inspection of canned food is a complex problem and although a vast amount of information is available for those who carry out the work, long experience is the most valuable aid. A team comprising the public health inspector, analyst, and bacteriologist can put up a very formidable barrier against the possibility of unsound or unwholesome canned food being passed on to the public.

Inspection technique includes some or all of the following examinations:

(1) External. Physical examination of exterior, noting general condition—rusts, leaks, damage, information from labels and codes (and any other relevant history ascertainable); occurrence and extent of visible bulging; integrity of body and end seams and of solder; indentations and perforations (examining closely for signs of minute damage such as 'pin-holing').

(2) Palpation. Resistance of ends to finger pressure.

(3) Auscultation (shaking). Particularly applicable to solid packs such as meat foods, where a sloppy or washy sound might indicate activity of 'sulphide spoilage' organisms or liquefaction of gelatine due to bacterial activity.

(4) Percussion (tapping). Comparing the dull note of sound cans with the resonance of doubtful ones. Loss of vacuum indicated by resonance may be confirmed by use of the vacuum gauge.

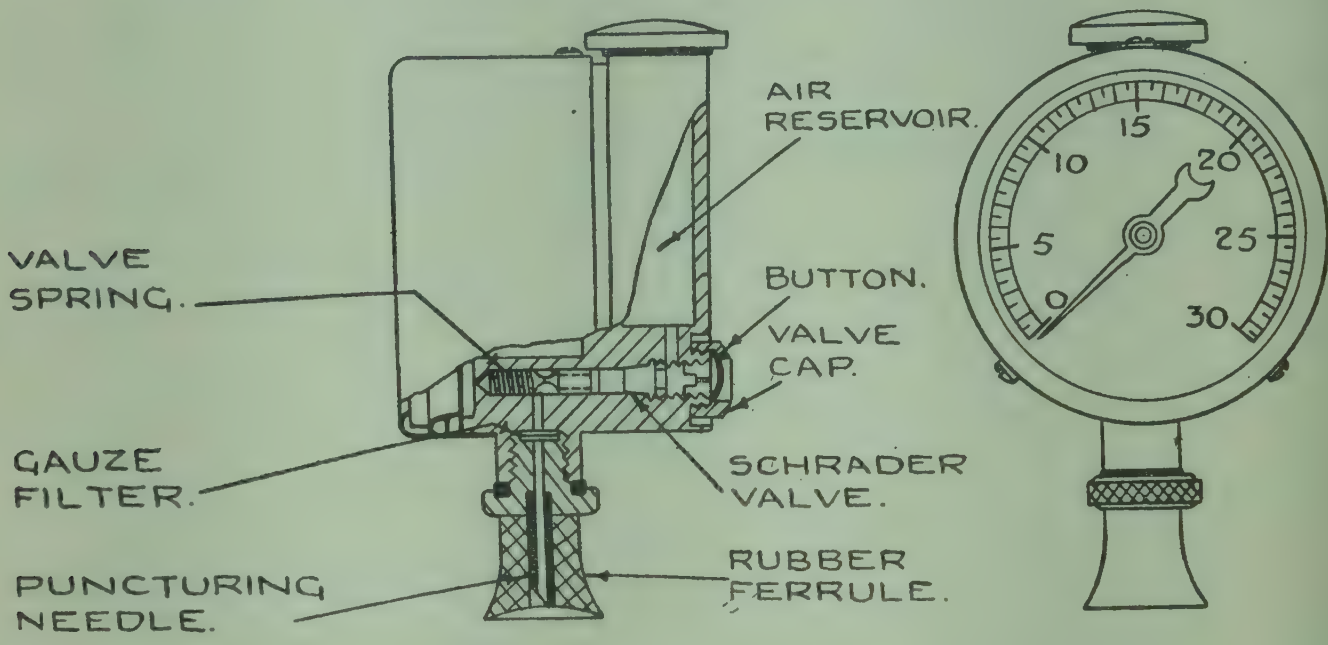

FIG. 15. CANNED PRODUCTS VACUUM GAUGE (courtesy *Budenberg Gauge Co. Ltd.*).

(5) Internal examination. Noting any escape of gas on opening, head-space, appearance of inner surface of container; colour, consistency, odour, taste of contents. Supplementary examinations may be necessary:

(*a*) Chemical, for *p*H values, contamination by metals, presence of preservatives, gas analysis.

(*b*) Bacteriological, for presence of specific organisms, aerobes, anaerobes, gas-producers, with particular reference to food-poisoning organisms and their toxins. Significance would also attach to types of organisms as indicators of efficiency of processing or post-process contamination.

Savage (1920) remarked, regarding reliability of visual examination of contents of opened cans:

Our results show definitely that a naked-eye examination is all that is required in ordinary cases. Of the samples examined, 49 appeared perfectly good in every way when opened. Of these, 42 were sterile and the remaining 7 only contained bacteria which were harmless, usually present in but scanty numbers and which were incapable when experimentally tested of initiating any decomposition changes in

artificially inoculated tins of corned meat. On the other hand, all the tins (with possibly one exception) which appeared unfit to the naked eye were infected with bacteria and mostly with varieties capable of decomposing the meat.

Jepsen (1957) remarks,

> When cultures from sterilised canned meats show definite growths of micro-organisms in significant numbers the product should always be regarded as suspect, no matter which are the species of organisms and whether organoleptic findings are positive or negative.

Jepsen in the same work gives a comprehensive account of the bacteriological examinations recommended and the interpretations to be placed on results, both for sterilized canned meats and for low-temperature heat-treated cured canned meats (hams, etc.). Hobbs (1956) recommends a sample of six unopened cans for bacteriological examination: if from a spoiled consignment, two blown and four normal.

Condition of Exterior

A normal can has a clean appearance, is free from rust, dirt and stains, has slightly concave ends, seams mechanically sound and is free from leaks. If the code marks can be deciphered it may be possible to establish the age of the sample. Absence or discoloration of labels should be viewed with suspicion; so should any inconsistency between age of cans and labels (new labels on old cans may suggest an attempt to lull suspicion over a doubtful consignment). The method of sealing should be examined and where there are additional soldered vent holes one should suspect resoldering after expulsion of gases of fermentation or decomposition. Where 'sanitary caps' have been used any soldered vent is suspicious, since the cap is crimped in a double seamer machine after exhausting. Although re-sealing of leaks by the contents (for example, by the fat of canned meat) may have occurred before contaminants have entered, any loss of vacuum is important and a favourable judgment in such cases should await a satisfactory laboratory report, supporting normal appearance of contents of a representative sample of affected cans.

Damage, due to ill-usage, usually leads to rejection of cans as unmarketable—certainly where seams have been strained such cans should be rejected. Signs of rust should be followed by a close inspection to ascertain if any have rusted through. For detection of pin-hole leakages through this cause it is useful to supplement

careful visual examination by palpation as the tiny holes are sometimes better heard than seen. In such cases it will be necessary to remove labels to assess correctly the effects of rust. Points at which adhesive has been used for affixing labels require detailed scrutiny for it has been found that unsuitable pastes may attack the can and cause pin-holing which the label covers up. Assessment of the effects of damage, etc., may necessitate the inspection of the packing cases and enquiries into modes of transit. Careless use of nails may result in one or more cans being punctured and the cases should be opened for visual examination.

Palpation

While blown cans can be identified visually, the early stages of loss of vacuum can best be ascertained by palpation. Whether heat or vacuum-exhausted, a sound can has both ends concave and the contents are closely adherent at all points of the container. When vacuum is lost the resultant loss of adhesion, in places or extensively, can be recognized by the fingers as a springiness. Occasionally excessive head space results in too great a vacuum and a collapsed can; equally exceptional overfilling, by reducing head space, may result in ends which are not concave, but judgment cannot be safely taken without examination of contents.

The most common defects of canned foods are 'blown' or 'swell' ('hard' when neither end is reducible, 'soft' when thumb pressure momentarily allows reduction), 'springer' (one end bulged and when pressed, opposite end bulges) and 'flipper' (one end bulges when opposite end struck). These conditions are usually brought about by bacterial spoilage, the 'flipper' representing an early stage of gas production, progressing through 'springer' and 'soft swell' to 'hard swell' or 'blown'. Acid and gas producing organisms commonly responsible have been identified by Jansen and Aschehoug (1951) with *B. subtilis*, *B. subtilis-B. pumilus* intermediates, and *B. macerans*. *Cl. perfringens* and *Cl. sporogenes* may be responsible for swelling of canned hams (Jepsen 1957, *op. cit*).

Rectangular containers for meats usually bulge at the sides rather than at the ends, and any doubt as to this change can best be resolved by comparison with a sound can. Occasionally an apparent 'swell' may be the result of damage to the side of the can, but this can be demonstrated by reducing the 'swell' when the corresponding indentation will temporarily assume normal contour. Such cases can only safely be approved when careful

examination of the can has shown no cracks, leaks, or defective seams.

'Slack caps'—cans in which an oversize cap has been used—have the appearance of 'blown' cans. Although this explanation may be correct, the majority of such cases are in the early stages of 'blowing' and favourable judgment can never be safely extended to any can which is in a physical condition of springiness consistent with gas production by micro-organisms.

Round cans in which fish are packed can be judged physically as other cans but thin-walled cans of large surface area, such as used for herrings and sardines, may normally exhibit slight springiness in a few sound samples, but extensive occurrence of this condition justifies rejection. Lang (1935) suggests that bad conductivity of oil in which fish are packed may lead to survival of organisms which in an aqueous medium are destroyed at normal processing temperatures. Before oval 'springers' are submitted for laboratory examination a simple field test may be performed by piercing a sample can under water. Unsound cans produce air (or gas) bubbles.

Head Space in Canned Fish

In South Africa (Dreosti and Roux, 1951) a head-space tester is used in the routine inspection of canned fish. Head space is defined as 'average depth of the surface of the contents below the underside of the lid . . . multiplied by the internal cross-sectional area of the can would then be the volume of the head-space'. Net head space should be less than $\frac{1}{4}$ inch for the following reasons:

(1) Attainment of maximum vacuum during steam exhaust.

(2) Inclusion of a minimum amount of O_2 in the cans at a given vacuum so as to avoid oxidation of oils and corrosion of cans, the most potent causes of which are high head space and low vacuum.

(3) Attainment of minimum effect on concavity of lids at a given vacuum and minimum number of springers under conditions of high temperature and pressure to which products might be subject.

(4) Minimum agitation of products in transit, thus avoiding turbidity of juice and spoiled appearance of contents. Pilchards, in particular, are subject to 'mush' when the head space exceeds $\frac{1}{4}$ inch and cans are vibrated continuously for some days, while those with low head space remain intact.

Head space less than $\frac{1}{8}$ inch does not allow for expansion of the product during processing.

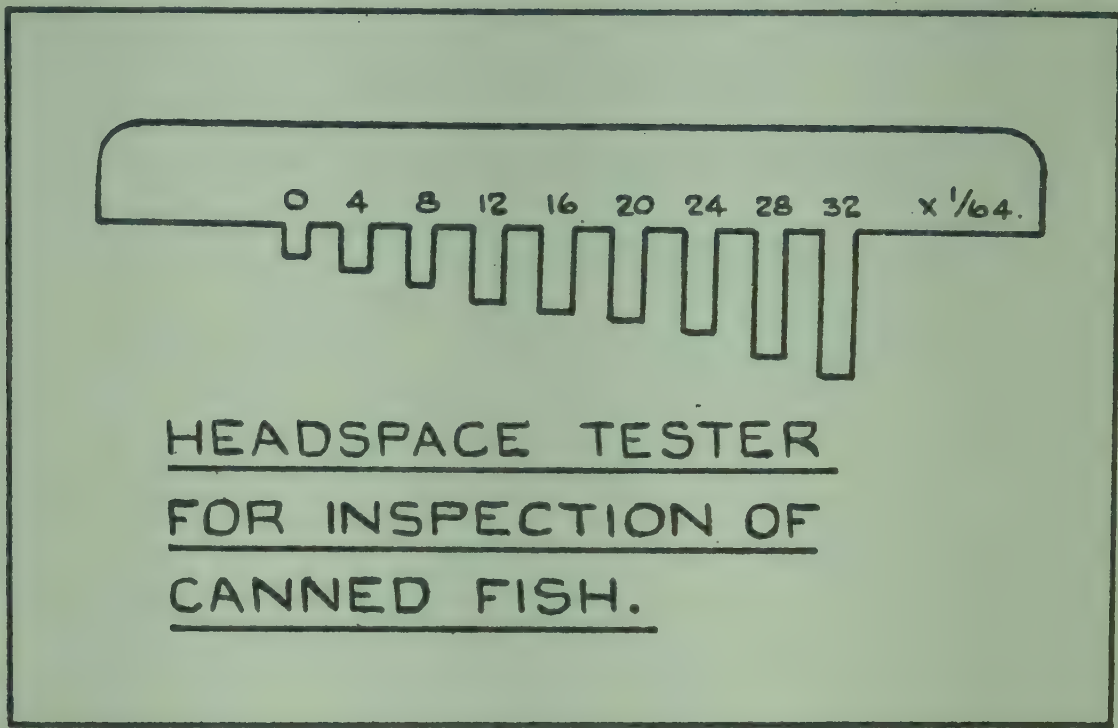

FIG. 16. HEADSPACE TESTER FOR INSPECTION OF CANNED FISH.

HYDROGEN SWELLS

Fruit of low acidity (pH 3·5–4·5)—raspberries, loganberries, blackberries, plums, cherries, etc.—and some vegetables, stored in a warm atmosphere, develop gas which causes cans to blow. The gas generated is hydrogen, as a result of electrolytic action set up by acid contents on exposed iron in the presence of tin. Atmospheric oxygen accelerates electrolysis and perforations may occur. Yeasts may also cause blowing. Hydrogen acts as a reducing agent on the fruit pigment, causing bleaching, and although contents in some cases may be sound, in others fruit may be soft and discoloured. Oxygen and anthocyanin (pigment of red fruits) act as depolarizers. Damage to the tinning during can manufacture is a contributory factor, by exposing iron. Lacquering also aids the establishment of an electro-couple by preventing the formation of stable compounds of tin which protect the iron. Corrosion is accelerated by the presence of sulphites but retarded by proteins. Large head space may result in retention of O_2 and will determine the stage at which H_2 production is indicated by the can.

Inefficient exhausting results in the retention of O_2 in the cans after sealing and oxygen may also gain access at the seams, especially if paper gaskets are used. Acidity of the fruit is highly significant—if less acid than pH 4 or of greater acidity than pH 3·5, hydrogen swells are unlikely to occur. Inefficient cooling of cans

after processing (especially corrosive effect of residual moisture) and high storage temperatures are also contributory factors.

REMEDIAL MEASURES

1. Careful attention to the technical and mechanical details, especially exhausting, sealing, and cooling.
2. Adjustment of *p*H value of syrup of 'low-acid' fruits by addition of 0·3–0·5 per cent citric acid.
3. Avoidance of sulphur compounds in sugar, since sulphites accelerate corrosion.
4. Use of beet-sugar as an inhibitor.
5. Addition of small quantities of inhibitors such as agar-agar.
6. Lacquering can rarely be omitted, since discoloration results from use of plain cans, but it must be carried out efficiently if detinning is not to be accelerated.
7. Cool storage.

Special Report No. 40 (Morris and Bryan, 1931) states:

> The attack on the metal by certain fruits may be so severe as to bring about perforation by small pinholes. Losses incurred in this way are not nearly so heavy, however, as those due to 'hydrogen swells' or 'springers' in which corrosion has resulted in the formation of sufficient hydrogen to cause the end of the can to bulge outwards.

Hydrogen swells may occur in any stage from 'flippers' to cans which bulge to the point of bursting and may affect any foods containing organic acids (fruit in syrup *p*H 3·5–4). Such cases may reveal large detinned areas. Although the type of food, internal appearance, and chemical analysis of gas may confirm 'hydrogen swell' it is difficult to extend a favourable judgment, since neither 'springer' nor 'swell' is accepted as a saleable can, whatever the cause, and as the elimination of other causes of gas formation can only be made on opening the cans, the further life of any contents adjudged sound would be very limited. Physical deterioration of contents as a sequel of the reducing properties of H_2 has already been noted and the large areas of detinning seen in many cases of hydrogen swell would lead to concern as to the possible tin content of the fruit. Where every factor was favourable, it would be possible to release certain fruits affected by hydrogen swell for purposes such as preserves manufacture under strict supervision.

SHAKING (AUSCULTATION)

The value of this operation depends on the contents of the can. It is, for instance, of little value in the case of fruits in syrup, and

of none in the examination of liquid or semi-liquid foods. It is of value, however, when examining canned fish or shell-fish, or solid packs such as meat. Held close to the ear, the sound can gives a firm solid thud. When a washy sound is heard, liquefaction of contents is likely, due to proteolytic organisms which cause spoilage with or without gas formation. Meat is usually affected by sulphide spoilage organisms, but occasionally the addition of water to prevent dryness of the product may give a misleading result. Liquefaction of gelatine may also be indicated, though high atmospheric temperatures may produce a similar result without the contents being unsound. In this case cooling will help to distinguish the sound can.

Percussion

The test is not universally applicable to all canned packs, but is useful with solid contents. When the can is tapped it emits a dull, solid, non-resonant sound, but if air or gas is present the sound will be tympanic and resonant. The sound may be confined to one part of the can or, where large amounts of air or gas are present, may be heard from all parts. A possible source of error arises from over-filled cans, which give a dull note consistent with soundness, while an under-filled can will give a resonant note, though sound, but here the weight shortage will usually be apparent.

Percussion is of little value in assessing soundness of canned fruit or vegetables, due to the increased head space. Leaks will give an 'air note' on percussion.

More accurate results than by percussion can be given by use of the improved vacuum gauge (F.I.R.A. gauge) developed by the British Food Manufacturing Industries Research Association in collaboration with the Budenberg Gauge Company.

Chemical Examination

Amounts and kinds of preservatives permitted in canned foods are, in this country, those laid down in the Public Health (Preservatives in Food) Regulations. Since there are varying national standards it may sometimes arise that imports are suspected of containing prohibited preservatives such as salicylic or boric acid. Antibiotics are similarly excluded at present, though other countries may permit them. Where prohibited ingredients are suspected the articles should be sent for chemical analysis. Suspected metallic contamination may similarly require submission of cans for chemical analysis.

Presence of toxins in canned foods cannot be detected by physical examination and even when suspected, as in food poisoning investigations, can only be surmised on the evidence of inoculation experiments in the bacteriological laboratory.

The presence of tin sulphide is sometimes noted on examination of contents of meat cans. Usually this occurs as a black staining of the interior of unlacquered cans and contents. Though unsightly this condition is not harmful—indeed the formation of tin sulphide protects the tinplate from further corrosive or electrolytic reaction.

Condensed Milks

Methods of inspection employed are similar to those used for other classes of canned food, but a detailed physical examination is necessary to detect bulges, leaks, excessive rust, and mishandling. Palpation will detect 'springiness' but there should be no sound on shaking, unless incompletely filled. High temperatures reached in processing evaporated milk mean that 'blowing' is almost invariably due to spore-forming organisms. Interiors of cans are often discoloured due to formation of tin sulphide, but there is no harmful effect.

Full cream and machine-skimmed milk usually spoil through activities of a non-sporing yeast. Moulds sometimes attack sweetened milk, coagulated dark patches appearing near the surface like 'buttons', due to coagulation of protein (casein). Caramelization of sugar may cause a discoloration which renders the contents unmarketable.

Curdiness of canned milk results from high acidity or rennet-producing organisms and is due to too low a heat-coagulation point. Heat-resisting enzymes may produce a rancid flavour in full-cream condensed milk, as a result of the release of free butyric acid when the fat is split. Contamination with micro-cocci during manufacture may produce thickening, especially of condensed milk consigned to the tropics. High acidity, high vacuum pan temperature, high sucrose concentration, high albumen content, or colloidal changes may be contributory factors. Except for 'blowing' and 'springiness' these defects are only ascertainable on opening the can and are individually or collectively responsible for the more limited store life of canned milks.

Dehydrated Canned Foods

It is recommended (Von Loesecke, 1943) that certain dehydrated foods, notably milk, meat, vegetables, be packed in cans, either

vacuum-sealed or charged with inert gas or CO_2. If the latter practice is followed internal gas pressure will cause bulging of can ends and such cans should not be regarded as 'swells' but accepted as sound. Where vacuum sealing is practised loss of vacuum will not be of the same significance, since contents are not normally sterile but should be judged in relation to physical evidence of spoilage, such as rancidity, excess moisture content, moulds, abnormal odour or flavour. Canned dehydrated foods do not enter into peacetime commerce to any significant extent.

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PART VI

Chapter XXV

LABORATORY INVESTIGATION OF FOOD-POISONING CASES

In investigating an outbreak of food poisoning, it must be remembered that different varieties of organisms may be responsible, and it is necessary to make cultures on various types of culture media or the causal organism may be missed. The main organisms to search for are:

(1) Salmonella group, the commonest of which are *Salm. typhi-murium*, *Salm. enteritidis* (including sub-strains), *Salm. thompson*, *Salm. newport*, and *Salm. dublin*.
(2) *Staphylococcus aureus*.
(3) Flexner and Sonne dysentery bacilli (only occasionally involved).
(4) *Clostridium welchii* (*perfringens*).
(5) *Clostridium botulinum*.

Salmonella Group

Material from the patient. Stools, vomit, and suspected food are emulsified, or macerated in saline solution, and are plated direct on to desoxycholate-citrate agar, or if this is not available, MacConkey's medium, and incubated overnight.

In addition, material is inoculated into enrichment media such as 'Selenite F' or tetrathionate broth. Several loopsful of material are added to the medium and incubated overnight.

With heavily infected material a liquid or semi solid-medium containing hydroquinone or cacotheline with brilliant green may be used to inhibit excessive growth of proteus.

The next day the desoxycholate medium plates are examined for colourless colonies, and if only few or none are present the Selenite F or tetrathionate broth cultures are inoculated on further desoxycholate-citrate plates. If colourless colonies are present, several colonies are picked off and each inoculated into 3 c.c. of broth. These are incubated for 3–4 hours and then from each is inoculated the following:

(*a*) Peptone water fermentation media with lactose.

(*b*) Peptone water fermentation media with glucose.
(*c*) Peptone water fermentation media with mannitol.
(*d*) Peptone water.
(*e*) MacConkey agar plate.

These are incubated overnight. The peptone water culture is tested for the presence of indol.

The usual reactions in sugar media by the Salmonella group of organisms are:

Medium	*Reaction*
Lactose, adonitol, and sucrose	No fermentation
Glucose, mannitol, sorbitol, and dulcitol	Acid and gas
Peptone water . . .	No production of indol

The MacConkey plate serves to check the purity of the organism isolated, in case the colourless colony picked off was contaminated with *B. coli*. The plate would show the contamination and enable a pure culture of the organism to be recovered from it. The culture in peptone water is essential to distinguish the paracolon bacillus which gives a colourless colony with the same sugar reactions as the salmonella group, but produces indol.

If the sugar reactions are correct, the broth culture from the colourless colony is tested for agglutination with a mixed salmonella agglutinating serum. If agglutination takes place the test is repeated, using sera from the individual members, and the organism definitely identified. If no agglutination occurs to the common members of the group the organism isolated must be sent to a reference laboratory for final identification. If no colourless colonies are sent in the original plates, then plates inoculated from the enrichment media should be examined. If colourless colonies are present they are picked off and examined as above.

If dysentery bacilli are present the reactions are:

Medium	*Reaction*
Lactose and sucrose . .	No fermentation
Glucose and mannitol . .	Acid but no gas
Peptone water . . .	Production of indol

The identity of the organism is ascertained by the use of the appropriate specific agglutinating serum.

Blood Culture. This should be carried out as a routine measure, and in some cases yields positive results with the salmonella group. 10 c.c. of blood are taken from a vein and inoculated into two blood culture bottles containing glucose broth and incubated at

37°C. A positive growth shows gram-negative bacilli which are tested for sugar reaction, indol production and agglutination as above.

Agglutination with Patient's Serum. The agglutination test is not applicable to the acute stage of the disease as agglutinins take 7–10 days to develop. It may be applied to convalescent or recovered cases (not previously diagnosed) mainly for purposes of epidemiological investigation. Blood (5 c.c.) is taken from a vein, allowed to clot, and the serum withdrawn. Tests are made with specific H suspensions of the commoner types. A reaction in a dilution of serum above 1 in 50 is accepted as significant. Previous typhoid-paratyphoid inoculation, however, will render agglutination results invalid.

Post-Mortem Material. Cultures are made from heart, blood, spleen, liver, and intestine.

Food. Portions of meat, sausage, etc., are emulsified or ground up in saline solution and desoxycholate-citrate plates, and enrichment medium inoculated as for faeces. Milk is centrifuged and the deposit inoculated on culture media as above. The examination of the cultures is the same as with faeces.

Identification of Types of the Salmonella Group

Salm. typhi-murium. Characteristics and reactions. The organisms occur as short, single (0·5 by 1·0 to 1·5 microns) motile rods which are gram-negative. Optimum temperature for growth 37°C., aerobic, facultatively anaerobic. Cultivated on gelatin, the colonies are small, circular and greyish to yellowish-brown in colour but no liquefaction occurs. Gelatin stab—flat surface growth, no liquefaction. On agar, the colonies are small, circular, greyish in colour, and entire to undulate. In broth culture there is turbidity but no production of indol. Litmus milk is at first turned slightly acid, but later becomes alkaline, no coagulation occurs. On potato, growth appears as a greyish-white streaky film. The organisms ferment glucose, fructose, galactose, arabinose, maltose, dextrin, mannitol, sorbitol, trehalose, ducitol, inositol, xylose, and rhamnose (variable). There is no action on lactose, sucrose, raffinose, inulin, salicin, or adonitol. Reduces trimethylamine oxide. Produces acid reaction in tartrate medium (variable). H_2S is produced. Antigenic structure: (I), IV, (V), XII, I; 1, 2, 3 . . .

Salm. enteritidis (Gaertner or Jena variety). The organism

appears as motile rods (0·6 to 0·7 by 2·0 to 3·0 microns) with peritrichous flagella. They are gram-negative, aerobic, and facultatively anaerobic. Occur singly or in pairs, and occasionally in short chains. Optimum temperature of growth 37°C. Cultivated on agar, colonies are circular, grey, translucent, granular, and entire. Gelatin stab—abundant growth, no liquefaction. Inoculated broth; turbidity with thin pellicle and greyish-white sediment. Indol not formed. Litmus milk: slightly acid, becoming alkaline, opalescent, translucent to yellowish-grey. Potato: abundant, moist, yellowish-brown to brown growth. Nitrites produced from nitrates. Sugar media: the organisms ferment glucose, fructose, galactose, mannose, arabinose, xylose, maltose, trehalose, dextrin, glycerol, mannitol, dulcitol, rhamnose and sorbitol. No acid and gas from lactose, sucrose, inulin, salicin, raffinose, adonitol, and inositol. Produces acid reaction in tartrate medium. Reduces trimethylamine oxide H_2S produced. Antigenic structure: (I), IX, XII: g, m –. *Salm. enteritidis* var. Danysz differs from *Salm. enteritidis* only in its negative action on glycerol in Stern's medium.

Although it may not be easy to determine that the organism responsible for food poisoning belongs to the salmonella group, it is often difficult to identify it precisely. It is such a highly specialized procedure that only a few laboratories can undertake it. In this country cultures should be sent to the 'Salmonella Reference Laboratory' at the Central Public Health Laboratory, Colindale Avenue, London, N.W.9.

It is not possible or desirable in a book such as this to give the minutiae of technical details, but the general underlying principles which illustrate the complexity of the work involved are as follows:

The organism is examined for the presence or absence of motility, and fermentation tests with a large and specified number of sugar media are carried out. These results are later correlated with the antigenic structure of the organism which is next investigated.

The first step is to identify the O or somatic antigen for which special antisera (made by injecting suspensions of different members of the sub-group into rabbits) are used.

Having ascertained the nature of the O antigen it is next determined whether the organism is in the specific or non-specific phase. If in the latter, then by selective cultivation a specific phase-colony is obtained and tested with specific-phase agglutination sera prepared against the types of salmonella in the sub-

group in which the O antigen (which was previously determined) belongs. It may thus be possible at this stage to identify salmonella types commonly met with.

If a rare or uncommon type is being examined, a complete range of salmonella-type sera may have to be used, including a serum prepared from the organism itself, and this may be involved and time-consuming.

The antigenic structure is complex. For example, a food-poisoning organism of the salmonella group may have an antigenic analysis of O antigen IX, XII and H antigen e.h.; 1·5.

The above description gives only a brief and rough outline of the investigations necessary, and is included here to indicate the many difficulties encountered before the final identification of a food-poisoning organism may be completed. It also shows the importance of maintaining a central reference laboratory where such high technical work is always available.

Staphylococcal Food Poisoning

Staphylococcus aureus (Rosenbach 1884). Characteristics and reactions: Spherical non-motile cells (0·8 to 0·9 microns in diam.), which occur singly, in pairs, short chains, irregular clusters or clumps. Gram-positive, non-sporing, aerobic, facultatively anaerobic. Optimum temperature for growth 37°C. Can be grown on all ordinary media. Cultivated on agar—colonies (2–4 mm. in diam.) are circular and opaque, having a smooth glistening surface and entire edge. Colour, golden-yellow to orange. Agar slope: abundant, opaque with smooth, flat, moist and glistening growth. Colour, yellow to orange in 24 hours' growth. MacConkey's Agar, characteristics: pinkish colonies, later turning red in colour. Broth turbid with a slight yellow ring on surface, powdery sediment which disintegrates on shaking. No production of indol. Gelatin stab—abundant growth, liquefaction occurs with yellowish pellicle and yellowish-orange sediment. Litmus milk acid with coagulation. Potato—slightly raised glistening yellowish-orange growth. Sugar media—acid reaction (no gas) in glucose, lactose, maltose, sucrose, mannitol, and glycerol. No reaction occurs in raffinose, salicin, or inulin. Nitrates reduced to nitrites. Slight H_2S reaction (varies). Coagulase produced which clots human or rabbit plasma. Starch not hydrolyzed.

Strains of staphylococcus vary considerably in the production of hemolysin, coagulase, and other metabolic products. Some

observers, however, are of opinion that power to coagulate citrated or oxalated blood plasma of rabbit or man is one of the most constant properties of pathogenic staphylococci. The work of Cowan, Christie, Keogh, Hobbs, Fisk, Wilson, and Atkinson has increased our knowledge of serological and bacteriophage types of coagulase-positive staphylococci. About 25 per cent of coagulase-positive strains of staphylococci can be identified by phage typing and about 98 per cent by serological typing. Recent studies in connection with the coagulase test suggest that when examining foods for the presence of food-poisoning staphylococci, an anti-coagulant, other than citrate, in the plasma be employed for the coagulase test. Any culture that requires more than three hours to clot citrated plasma should be regarded with suspicion.

Clostridium welchii (*perfringens*).

Characteristics and reactions: A gram-positive non-motile bacillus. Rods short, thick (1·0 to 1·5 by 4·0 to 6·0 microns.) with square or rounded ends. Occur singly or in pairs, but sometimes in chains. Spores oval, encapsulated, and subterminal. Optimum growth 35°C. to 37°C. (anaerobic). Grows well on glucose agar. Agar surface, raised circular colonies, with opaque centre. Broth turbid, peptolytic with sediment. Indol not formed. Litmus milk acid, coagulated, with profuse production of gas. The culture has a sour butyric-acid odour. Gelatin is liquefied. Blood serum not liquefied. Cooked meat medium, pink with gas. Nitrates produced from nitrites. Acid and gas produced in glucose, fructose, lactose, sucrose, maltose, zylose, dextrose, trehalose, raffinose, starch. Salicin and inulin are rarely fermented. Mannitol and dulcitol not fermented.

Suspected food material, vomit, and faeces are inoculated on blood agar plates and incubated at 37°C. When *Staphylococcus aureus* is responsible a heavy and almost pure growth is obtained from food or vomit, which is haemolytic and coagulase positive.

The coagulase test is carried out as follows. Citrated human plasma is diluted 1 in 10 in normal saline, and about 0·5 c.c. of diluted plasma placed in each of two test tubes. To one tube is added 5 drops of an overnight broth culture of the staphylococcus. Both tubes are then incubated at 37°C. Clotting usually occurs within one hour. The uninoculated tube acts as the control and should show no clotting.

The clinical symptoms are caused by a staphylococcal entero-

toxin which is resistant to heat. Broth cultures from staphylococci may be grown in broth, filtered through a Seitz or Berkefeld filter, heated to 56°C. for 20 minutes to remove haemolysin, and the filtrate injected intraperitoneally into a kitten, when symptoms of gastro-enteritis (vomiting and diarrhoea) appear in a few hours. The phage testing of staphylococci isolated from food and suspected carriers is often useful in epidemiological investigation.

Clostridium botulinum

Characteristic sand reactions. Types A and B can be cultivated on all ordinary media under strictly anaerobic conditions. Optimum temperature 35°C., but grows well at 20°–30°C. A powerful exotoxin is produced which is specific to each type. Organisms occur as fairly large, straight, thick rods (0·5 to 0·8 by 3·0 to 8·0 microns) with slightly rounded ends, arranged singly or in pairs and sometimes chains. Motile by 4 to 8 flagellae. The spores are oval and situated on or near the ends (sometimes free) which are bulging and wider than the organism. Gram-positive especially in young cultures.

Types A and B are proteolytic (types C and D digest gelatin only) and digest coagulated white of egg.

Growth on agar plate greyish-yellow and translucent with granular surface. Agar stab: a white line of growth with short lateral radiations; gas is produced. Gelatin—complete liquefaction. Broth culture—abundant growth, liquid turbid with powdery or granular deposit. Rancid odour evolved. (Types C, D, and E give slight turbidity with flaky deposit.) No production of indol.

Sugar medium, type A, ferments (acid and gas) glucose, maltose, and salicin. Types B and C do not ferment salicin. Types A and B ferment glycerol but type C does not. Litmus milk—litmus reduced, reaction alkaline. Casein precipitated and digested. Meat medium (cooked): heavy growth with production of gas and putrid smell. The fluid is dark and turbid with blackened meat sediment. No digestion by types C, D, or E.

Demonstration of Toxin-Suspected Food. A suspension of the food is made in saline and injected intraperitoneally into three mice. Two of the mice are given subcutaneous injections of botulinus antitoxin A and B respectively a few hours before the test. The death of the control mouse and one of the antitoxin injected mice, indicates the presence of botulinus toxin corresponding to the type of antitoxin administered to the surviving mouse.

Demonstration of the Organism in Food. The food material is macerated in sterile saline solution and heated at 65°C. for 30 minutes to destroy non-sporing organisms. Cultures are made in glucose broth and cooked meat medium, and incubated anaerobically for 7–10 days at 37°C. The culture is then filtered through a Seitz or Berkefeld filter and the filtrate injected into three mice as above. To isolate the bacillus the cultures are inoculated into deep agar stabs and on blood agar plates and incubated anaerobically. Single colonies are picked off and tested for toxin production. Similar methods are used in investigating stomach contents and faeces. With post-mortem material, liver, spleen, and intestinal contents are examined.

Summary of Procedure of the Bacteriological Examination of Material from a Case of Food Poisoning

With each sample of material macerate or emulsify portions in saline, then

1. Inoculate two petri plates of desoxycholate-citrate-agar medium.
2. Inoculate enrichment media, either 'Selenite F' or tetrathionate broth, or both.
3. Inoculate two blood agar plates.
4. Heat saline suspensions of food or stomach contents to 65°C. for 30 minutes. Inoculate two bottles cooked meat medium, and two of glucose broth, which are incubated anaerobically.
5. If botulism is suspected carry out mouse test for the presence of toxin.

Examine the inoculated culture media as described previously.

It is important to carry out the bacteriological examinations of material as soon as possible after a case of food poisoning has been notified. The bacteriologist, if immediately available, should collect his own material, or a special messenger sent to the laboratory without delay.

If material has to be sent any distance, or by post, or if delay in dealing with the specimens arises, a portion of each specimen should be placed in 10 c.c. of 30 per cent glycerol in 0·6 per cent salt solution. This will prevent overgrowth by putrefactive bacteria and greatly assist in the isolation of salmonella organisms. The remainder of the material should be kept cool and, where possible, in a refrigerator.

Culture Media

CULTURE MEDIUM FOR THE SELECTIVE ISOLATION OF STAPHYLOCOCCI (Chapman-Stone)

Water	1,000 ml.
d-Mannitol (Difco)	10 gm.
Bacto tryptone	10 gm.
Bacto-agar	15 gm.
Sodium chloride, Merck reagent	55 gm.
Bacto yeast extract	2·5 gm.
K_2HPO_4, anhydrous, Merck reagent	5 gm.
Bacto-gelatin	30 gm.
Ammonium sulfate, Baker analysed, A.C.S. pyridine-free	75 gm.
Sodium hydroxide, 10 per cent.	6 ml.

Sterilize 10 minutes at 15 lb. pressure. While still hot, shake thoroughly to disperse the precipitate and pour 40 plates from each liter. If the plates are poured when the medium is too cool, it will be lumpy and there will be pitting from air bubbles. Do not let the flask stand on a cool surface while it is cooling or the adjacent medium will solidify, making the medium lumpy. When properly prepared, the medium is uniformly opaque and free from coarse particles, lumps, and pits. It is translucent when warm but becomes opaque on cooling to about 25° to 30°C. (77° to 86°F.).

Chapman (1948) recommends the above medium for the rapid routine testing of foods thought to be the cause of food poisoning.

To use the Chapman-Stone medium in investigating a suspected outbreak of staphylococcus food poisoning, plate 0·01 ml. of several decimal dilutions of suspension of the suspected food and 0·10 ml. of a heavy fecal suspension, and rub the nose, throat, or other swab over the surface of the medium and spread the inocula with a glass spreader. Incubate the plates at 30°C. for exactly 48 hours. If the plates cannot be examined immediately they should be stored in sealed cans in the refrigerator, under which conditions they will remain suitable for testing for several days. Yellow or orange colonies that are surrounded by a clear zone probably are food-poisoning staphylococci.

On this medium, staphylococci produce deeper pigment than by previous methods; they coagulate blood faster; they show the Stone reaction without having to flood the plate with ammonium sulfate solution; and fermentation of mannitol is determined, all on the original isolation plate.

Heat to dissolve, adjust to *p*H 9·2 using thymol blue, add 25 gm. agar, dissolve in steamer, distribute in screw-capped bottles

MEDIUM FOR THE ISOLATION OF STAPHYLOCOCCUS AUREUS FROM HEAVILY CONTAMINATED MATERIAL

(Ludlam, 1949)

Mannitol	10 gm.
Dipotassium hydrogen phosphate (anhyd)	5 gm.
Lithium chloride	5 gm.
Lab-Lemco	10 gm.
Bacto peptone (or Evans Bacteriological Peptone)	10 gm.
Distilled water	1 litre

in 100 ml. amounts, and autoclave at 15 lb. for 20 minutes. Before use add sterile 1 in 400 potassium tellurite solution to the melted base to give a final concentration of 1 in 20,000. After 48 hours incubation at 37°C. colonies of *Staph. aureus* are 3–4 mm. in diameter, usually with a narrow pale border, sometimes showing slight golden pigmentation. *Staph. albus* either fails to grow or produce smaller pale colonies.

DESOXYCHOLATE-CITRATE-AGAR

(Modification by Hynes of Leifson's medium)

(Mackie and McCartney, 1953)

Medium for the Isolation of Organisms of the Salmonella and Dysentery groups.

Agar	22·5 gm.
Lab-Lemco	5·0 gm.
Peptone (Difco proteose or Evans)	5·0 gm.
Lactose	10·0 gm.
Sodium citrate	8·5 gm.
Sodium thiosulphate	8·5 gm.
Ferric citrate	1·0 gm.
Sodium desoxycholate	5·0 gm.
Neutral red (as indicator)	
Tap water to 1 litre	

It is convenient to make up a four-litres batch as follows. Dissolve 20 gm. Lab-Lemco in 200 ml. water over the flame; make just alkaline to phenolphthalein with 50 per cent NaOH, boil and filter. Adjust the *p*H to 7·4, make up the volume to 200 ml. and add 20 gm. peptone. Dissolve 90 gm. agar in 3,700 ml. tap water by one hour's steaming. Filter the agar, add the Lab-Lemco peptone solution and mix. Add 5 ml. 2 per cent solution of neutral red and 40 gm. lactose, and mix. Bottle in accurate 100 ml. lots, and sterilize by free steam for one hour and then at 5 lb. pressure for ten minutes.

Solution A

Sodium citrate (Analar, $Na_3C_6H_5O_7$, $2H_2O$)	17 gm.
Sodium thiosulphate (Analar, $Na_2S_2O_3$, $5H_2O$)	17 gm.
Ferric citrate (scales)	2 gm.
Distilled water	100 ml.

Dissolve by heat or by standing at room temperature for 2 days.

Solution B

Sodium desoxycholate	10 gm.
Distilled water	100 ml.

Sterilize these solutions at 60°C. for one hour.

For use, melt 100 ml. of the agar base, and add 5 ml. each of solutions A and B in this order, using separate pipettes and mixing well between. Pour plates *immediately* and dry surface.

The medium should be poured and cooled as soon as possible after the addition of the desoxycholate, otherwise it tends to become very soft. The desoxycholate must be pure and samples tested with known positive specimens before purchase is made.

The medium is pale pink in colour and should be quite clear. The colonies of pathogens are colourless.

MACCONKEY'S BILE-SALT NEUTRAL RED LACTOSE AGAR (Mackie and McCartney, 1953)

Dissolve by heat in tap water, peptone, 2 per cent, and sodium taurocholate (commercial), 0·5 per cent. Then add 2 per cent agar and dissolve in the steamer or autoclave. Clear with white of egg and filter. (Large quantities may be filtered through paper pulp in the same way as agar.) Add a sufficient amount (about 0·7 ml. per 100 ml.) of a freshly prepared 1 per cent watery solution of neutral red to give the medium a distinct reddish-brown colour. If the medium is acid, and assumes a rose-pink colour, add caustic soda solution until the colour becomes definitely reddish-brown. (It is preferable to adjust the reaction beforehand to *p*H 7·5 which gives

DRIGALSKI AGAR (modified)

Liebig's extract agar (2·2 per cent.)	1,000 ml.
Lactose solution (33 per cent.)	30 ml.
Sodium thiosulphate	1 gm.
Bromthymolblue solution (1 : 500)	40 ml.
Crystal violet solution (1 : 1,000)	5 ml.
(*p*H 7·7)	

BRILLIANT GREEN AGAR (Kristensen, Lester, and Jurgens) (modified)

Peptone	10 gm.
Liebig's meat extract	5 gm.
NaCl	5 gm.
Lactose	15 gm.
Tap water	1,000 ml.
0·5 per cent. Brilliant green solution	2 ml.
Phenol red solution	40 ml.
(Phenol red solution: phenol red 1 gram—40 ml. 1/10 normal NaOH—460 ml. distilled water)	
Agar	26 gm.
(*p*H 7·0–7·2)	

SELENITE F

Enrichment medium

(Mackie and McCartney, 1953)

Sodium acid selenite	4 gm.
Peptone	5 gm.
Lactose	4 gm.
Disodium hydrogen phosphate (Na_2HPO_4)	9·5 gm.
Sodium dihydrogen phosphate (NaH_2PO_4)	0·5 gm.
Distilled water	1 litre

the correct colour with neutral red.) Sterilize the medium in the steamer and when cool add 1 per cent lactose (previously sterilized separately in a 10 per cent watery solution). Sterilize the completed medium as in the case of other sugar media.

Distribute the yellowish solution in 10 ml. amounts in screw-capped bottles. Sterilize by steaming at 100°C. for thirty minutes (excessive heat is detrimental and autoclaving must not be used). A slight amount of red precipitate may form but this does not interfere with the action of the medium. The pH of the medium should be 7·1, and the phosphates may be varied slightly if necessary to attain this.

For use, a bottle of the medium is inoculated with two or three large loopfuls of faeces and incubated overnight. Sub-inoculations are made on desoxycholate-citrate medium.

Harvey and Scott Thomson (1953), as the result of investigations using Selenite broth and Wilson and Darling's (1918) Brilliant green MacConkey's medium, regard 43°C. (instead of 37°C.) as the optimum temperature for the incubation and isolation of salmonellae (except *Salm. typhi*). Harvey (1956) recommends the following selective medium for the routine isolation of members of the salmonella group (except *Salm. typhi*): 1/25,000 Brilliant green added to MacConkey's medium, pH when cold about 7·4.

TETRATHIONATE BROTH

(Mackie and McCartney, 1953)

To 90 ml. of ordinary broth add 2·5 gm. of chalk (previously autoclaved at 10 lb. pressure and then dried) and sterilized the mixture by steaming for half an hour. Add to the chalk-broth 10 ml. of a 60 per cent solution of crystallized sodium thiosulphate solution (sterilized by steaming for thirty minutes) and 2 ml. of iodine solution (prepared by grinding in a mortar 6 gm. of iodine and 5 gm. of potassium iodide and dissolving in 20 ml. distilled water). Distribute in 10 ml. amounts in tubes or screw-capped bottles. A tube or bottle of the medium is inoculated with faeces

and incubated for eighteen to twenty-four hours when a sub-inoculation is made on MacConkey's or desoxycholate-citrate medium.

As tetrathionate broth does not keep for more than several weeks it is convenient to prepare the solutions and make up the medium as required.

A. Sodium thiosulphate. Weigh out 24·8 gm. of $Na_2S_2O_3$, $5H_2O$ and dissolve in distilled water to make a final volume of exactly 100 ml. This gives a M/1 solution. Sterilize by steaming.

B. Iodine. To about 50 ml. of warm distilled water add 20 gm. of potassium iodide. Add 12·7 gm. of iodine and make up to a final volume of 100 ml. This gives a normal or M/2 solution.

To prepare the tetrathionate broth (100 ml. amount) add 2·5 gm. of chalk to 78 ml. nutrient broth and sterilize by steaming. When cool, add 15 ml. of the thiosulphate solution, 4 ml. of the iodine solution, and 3 ml. of 0·02 per cent solution of phenol red in 20 per cent alcohol as indicator. Distribute in 10 ml. amounts in bottles. Keep in the refrigerator and the medium will last several weeks.

Williams Smith (1952) investigated the evaluation of culture media for the isolation of Salmonella organisms from the faeces of man and other animals and birds. He found that selenite and tetrathionate media were greatly superior to other media for the recovery of these organisms from faeces.

WILSON AND BLAIR'S BISMUTH SULPHITE MEDIUM
(Mackie and McCartney, 1953)

Prepare a stock bismuth-sulphite-glucose-phosphate mixture as follows:

Dissolve 30 gm. bismuth-ammonio-citrate scales in 250 ml. boiling distilled water. Add to this a solution obtained by boiling 100 gm. anhydrous sodium sulphite in 500 ml. distilled water, and then while the mixture is boiling add 100 gm. sodium phosphate crystals (Na_2HPO_4, $12H_2O$). To the bismuth-sulphite-phosphate mixture when cool add a solution of glucose obtained by dissolving 50 gm. of commercial glucose in 250 ml. boiling distilled water. This mixture will keep for months.

Prepare an iron-citrate-brilliant-green mixture consisting of

1 per cent solution of iron citrate scales (ferric citrate scales) in distilled water	200 ml.
1 per cent brilliant green in distilled water . .	25 ml.

This mixture will keep for months.

Make up the medium as follows:

Nutrient agar, 3 per cent (melted and cooled to 60°C.)	100 ml.
Stock bismuth-sulphite-phosphate-glucose mixture	20 ml.
Iron-citrate-brilliant-green mixture	4·5 ml.

Pour into petri dishes.

WILSON AND BLAIR'S BISMUTH SULPHITE MEDIUM

(Zagreb modification by Cernozubov, Filipovic, Herrmann, and Staveł, 1944)

The formula is as follows:

(1) 20 per cent dextrose in distilled water. Boil and restore to original volume.

(2) Brilliant green Grubler in distilled water 1 per cent. Shake till dissolved.

(3) 20 per cent Na_2HPO_4 (*puriss. sicc.*) in distilled water. Boil and restore to original volume.

(4) 20 per cent Na_2SO_3 (*puriss. sicc.*) in distilled water. Boil and restore to original volume.

(5) 8 per cent $FeSO_4$ in distilled water. Do not boil. Add $2N/H_2SO_4$, 0·5 c.c. to each 100 c.c.

(6) 12 per cent bismuth ammonium citrate (Merck or Grubier). Do not boil.

To 1 litre of sterile melted 3 per cent agar add, in order:

(1) 50 c.c., (2) 5 c.c., (3) 50 c.c., (4) 100 c.c., (5) 10 c.c., (6) 50 c.c. Mix well and pour plates at once.

HOUSTON'S MEDIUM (1913)
(Mixed sugar gelatine)

For the isolation of salmonellae and other pathogenic organisms

Peptone (Evans)	20 gm.
Gelatine	75 gm.
Water	1,000 ml.

The gelatine medium is prepared and adjusted to *p*H 7·4.

To every 1,000 ml. of above gelatine medium is added:

Glycerol	2 ml.
Raffinose	2 gm.
Sucrose	2 gm.
Adonitol	2 gm.
Salicin	2 gm.
Lactose	2 gm.
Litmus 6 per cent aqua soln	15 ml.

The medium is distributed in $6'' \times \frac{1}{2}'$ test tubes as for stab cultures.

A SELECTIVE MEDIUM FOR THE ISOLATION OF SALMONELLA BACILLI FROM HEAVILY CONTAMINATED MATERIAL

Cacotheline-Brilliant Green Broth

(Ewart, Jones, and Handley, 1945)

The liquid and semi-solid media are easily prepared by the addition of appropriate amounts of stock solutions to ordinary infusion broth. Although both hydroquinone and cacotheline appear to be reasonably stable in aqueous solution, it is preferable to renew the stock solutions every 2 weeks. Broth and soft agar to which hydroquinone or cacotheline has been added should be used within 3 days of preparation. Since cacotheline does not appear to exert an inhibitory effect on those strains of Sal. *choleraesuis* sensitive to hydroquinone, and yet is equally inhibitory to *Proteus* strains and other unwanted organisms, we regard this substance as preferable for routine purposes. The supply of cacotheline is, for the present, somewhat restricted, though it may be obtained in small quantities. Where large volumes of media are employed we therefore have used hydroquinone as the active reagent, reserving the use of cacotheline for the Craigie tubes. Little appears to be known of the physical and chemical properties of cacotheline or of its molecular structure, it is much less soluble than hydroquinone, and concentrated solutions are not readily obtained. Two methods of preparation have been devised and are described below; both yield satisfactory media, but the use of the more stable stock solution (Method B) will probably be found more convenient.

Method A.—Cacotheline is dissolved directly in infusion broth to give a concentration of 1/500. This solution is then diluted with infusion broth to give a final concentration of 1/1,500, and the appropriate amount of a solution of brilliant green added to complete. In practice 0·1 g. cacotheline (B.D.H. Spot Test Reagent) is added to 50·0 ml. sterile beef infusion broth in a flat-bottomed flask or large boiling tube. Solution is effected by heating gradually, with constant shaking, in a water-bath until boiling-point is reached. Boiling is continued for 2 minutes, when solution should be complete. During the heating, the colour of the medium becomes dark brown and on cooling changes to deep olive green. To prepare 60 ml. of the complete medium mix the following in a sterile container:—

20·0 ml. of the heated 1/500 solution in infusion broth.

38·8 ml. of sterile broth.

1·2 ml. of a sterile 1/1,000 aqueous solution of brilliant green. The medium will be found satisfactory in use for at least 3 days after preparation.

Method B.—A 1/100 solution of cacotheline is prepared by dissolving 0·2 g. in 10 ml. of accurately prepared N/10 NaOH in the cold. When solution is complete 10 ml. of accurately prepared N/10 H_2SO_4 is added. As the acid is run in, the cacotheline is precipitated in a fine state of division but redissolves when the process is completed.

A perfectly clear solution is obtained on warming slightly. 60 ml. of the final medium are obtained by mixing the following:—

4·0 ml. of the 1/100 cacotheline solution.

54·8 ml. of sterile infusion broth.

1·2 ml. of sterile 1/1,000 brilliant green solution.

This medium has similar keeping properties to that prepared by Method A. The substitution of 9 ml. of 2 per cent. nutrient agar for a similar volume of infusion broth gives a satisfactory soft agar medium for use in the Craigie tubes.

Hydroquinone-Brilliant Green Broth.—A 1/80 solution of pure hydroquinone in distilled water is prepared and heated to boiling for 2 minutes in a water-bath, preferably in a stoppered tube. 100 ml. of the medium are obtained by mixing the following solutions in a sterile container:—

2 ml. of the 1/80 hydroquinone solution.

96 ml. of sterile infusion broth.

2 ml. of a sterile 1/1,000 solution of brilliant green.

A soft agar medium is prepared by substituting 15 ml. of 2 per cent nutrient agar for a similar volume of broth.

MEDIUM FOR THE ISOLATION OF ORGANISMS OF THE SALMONELLA GROUP
(Soltys, 1951)

3 per cent nutrient agar	700·0 ml.
Sodium taurocholate	5·0 gm.
Neutral red	0·05 gm.
Brilliant green	0·5 gm.
Acid fuchsin	0·5 gm.
Boiled milk	300·0 ml.

The basic medium is 3 per cent nutrient agar containing 0·5 per cent of sodium taurocholate adjusted to *p*H 7·2 and kept in 100 ml. bottles each containing 70 ml. of medium. When preparing plates, melt the agar and to each 70 ml. add 0·5 ml. of 10 per cent watery solution of acid fuchsin, 0·05 ml. of 10 per cent watery solution of neutral red, 0·5 ml. of 10 per cent alcoholic solution of Brilliant green, and 30 ml. of milk. All components should be sterilized before use and mixed thoroughly before pouring into petri plates. The medium is blue. Colonies of lactose-fermenting organisms have a deep indigo colour which does not diffuse into the medium, while non-lactose fermenters are white. Proteus is partially, and gram-positive bacteria are completely suppressed.

LIQUID UREA MEDIUM FOR IDENTIFYING SALMONELLA AND SHIGELLA IN FAECES AND RECTAL SWABS
(Modified Christensen's Urea Medium Base)
(Maslen 1952)

Difco bacto protease peptone	1 gm.
Dextrose	1 gm.
Potassium dihydrogen phosphate (KH_2PO_4)	2 gm.
Sodium chloride	5 gm.
Phenol red (0·04 per cent)	10 ml.
Distilled water	1,000 ml.

Steam until dissolved (10 minutes) and adjust to pH 6·7–8. Bottle in 180 ml. amounts in 250 ml. screw-capped bottles and sterilize by autoclaving for 10 minutes at 15 lb. (1 kg. per cm.^{2}) pressure. Allow the medium to cool. Prepare a 20 per cent solution of urea and sterilize by passing through a Seitz filter. Add 10 ml. of urea solution to every 90 ml. of base. Under sterile conditions pipette 1 ml. amounts into sterile bijou bottles. Incubate at 37°C. for 24 hours as a sterility test.

CULTURE MEDIUM FOR THE ENRICHMENT OF SALMONELLAE AND GROWTH INHIBITION OF PROTEUS

(Rappaport, Skariton-Loewenthal, and Olitzki)
(1953)

Bacto peptone (Difco)	10·0 gm.
KH_2PO_4	1·6 gm.
NaCl	8·0 gm.
Distilled water pH 6	1,000 ml.

Distribute in amounts of 5 ml. per test tube in 4 series. The following thiosulphate solutions are made:

Ca thiosulphate

$Na_2S_2O_3$	40 gm.
$CaCl_2$	18 gm.
Distilled water	100 ml.

Mg thiosulphate

$Na_2S_2O_3$	16 gm.
$MgCl_2$	14 gm.
Distilled water	100 ml.

These are heated together in the open autoclave for 30 minutes. To the series of tubes of medium are added 0·5 or 0·7 ml. of Ca thiosulphate solution or 0·9 or 1·3 ml. of Mg thiosulphate solution. Emulsion of faeces is inoculated into these media, and it is found that proteus is inhibited and the growth of strains of salmonella enriched.

MODIFIED TETRATHIONATE ENRICHMENT MEDIUM FOR CERTAIN SALMONELLAE

(Rappaport, Hirschberg, and Konforti)
(1956)

A modified tetrathionate broth medium is prepared from the following:

Bacto-Tryptose	5 gm.
$CaCO_3$	10 gm.
Bile salt (Difco)	1 gm.
Sodium thiosulphate	30 gm.

The dry ingredients are mixed in powder form in a mortar and then in a bottle on a shaker and stored. For use 4·6 gm. of the mixture are dissolved in 100 ml. distilled water and to this is added 3 ml. of a 0·4 per cent solution of malachite green and 4 ml.

of Lugol's iodine (25 gm. iodine and 20 gm. KI in 100 ml. distilled water). The medium is distributed in volumes of 5 ml. and is then ready for use. It must not be heated. The best results are obtained by emulsifying faeces in saline to give a 1 in 1,000 dilution and adding 3–4 drops of this to the medium. After incubation for 16–18 hours sub-cultures are made on to solid media (SS agar). Medium should *not* be used for isolation of *Salm. typhi* or *Salm. paratyphi* A.

FERROCHLORIDE GELATIN

(For H_2S formation and gelatin liquefaction)

Liebig's meat extract	7·5 gm.
Peptone (Parke, Davis)	25 gm.
NaCl	5 gm.
Tap water	1,000 ml.
Gelatin	100 gm.
10 per cent $FeCl_2$ ($4H_2O$)	5 ml.

The *p*H of the medium should be 7·6. Tube in narrow tubes (about 1 cm. diam.). Inoculate by making a stab culture and observe for at least 60 days at about 22°C.

STERN'S GLYCEROL FUCHSIN BROTH

Solution A

Liebig's meat extract	10 gm.
Peptone	20 gm.
Tap water	1,000 ml.

*p*H 8·0.

Solution B

Saturated alcoholic fuchsin solution . . . (10 per cent)

Solution C

Fresh aqueous 6 per cent anhydrous sodium sulphite solution.

Mix.:

Solution A	100·0 ml.
Solution B	0·2 ml.
Solution C	2·0 ml.
Glycerin	1·0 gm.

RINGER SOLUTION

Sodium chloride	9 gm.
Calcium chloride	0·25 gm.
Potassium chloride	0·42 gm.
Water	1,000 ml.

SPECIAL MEDIUM FOR FERMENTATION REACTIONS
(Braun and Ozak, 1946)

It is claimed that the fermentation reactions of salmonella species can be determined with greater accuracy, simplicity, and economy by the use of a synthetic medium in which ammonium salts are the source of nitrogen.

Formula:

$(NH_4)\ H_2PO_4$	1·15 gm.
Na_2SO_4	0·5 gm.
$MgSO_4$	0·01 gm.
Aq Dist	100·0 ml.

The solution is adjusted to *p*H 7·2–7·4, then sterilized and 0·2 per cent of the substate added. The substates are arabinose, rhamnose, dulcitol, glucose, lactose, sodium lactate, and sodium citrate. The medium with sodium citrate, a source of carbon, was used to test ammonia utilization. Tubes 16 × 8 were inoculated with a dilution of an agar suspension overlaid with sterile paraffin and incubated for 7–10 days. Suitable controls are necessary.

PEPTONE WATER

Peptone 1 per cent.

Sodium chloride 0·5 per cent.

Dissolve in warm water and afterwards filter. Sterilize by autoclaving.

EHRLICH'S ROSINDOLE REAGENT

Para-dimethyl-amino-benzaldehyde	4gm.
Absolute alcohol	380 c.c.
Pure hydrochloric acid	80 c.c.

Add an equal quantity of the reagent to a two-days culture of the organism in peptone water. A rose colour develops in the presence of indole.

RUY'S MEDIUM

To peptone broth add brilliant green 1 in 100,000 and 2 per cent Esbach's reagent (1 per cent picric acid and 2 per cent citric acid). A range of serial dilutions of brilliant green from 1 to 10,000 to 1 in 100,000 is an advantage.

Note for preparation of sugar media and blood agar see *Handbook of Practical Bacteriology*, Mackie and McCartney, 1953.

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APPENDIX I

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Food Poisoning

Steps to be taken in England and Wales by Medical Officers of Health in the Investigation and Control of Food Poisoning

Memorandum 188/Med. on Food Poisoning was first issued in 1935 and extensively revised in 1949. Between the dates of original publication and first revision there had been two developments of considerable importance in the better ascertainment and more precise knowledge of disease conveyed by food, namely, the notification of food poisoning under Section 17 of the Food and Drugs Act, 1938, now Section 26 of the Food and Drugs Act, 1955, and the establishment of the Public Health Laboratory Service, administered by the Medical Research Council on behalf of the Ministry of Health. Since 1949 the Public Health (Infectious Diseases) Regulations, 1953, Part 3, have given Local Authorities additional powers in regard to cases and carriers of certain food poisoning organisms, and a considerable amount of information has been collected on incidence and causes. The reported incidence of food poisoning shows no sign of decreasing, and there were 6,898 outbreaks (including family outbreaks) between 1950 and 1956: the causal food was named in less than a third of these outbreaks, and the organism was identified in approximately only two-thirds of those outbreaks in which a particular food was incriminated. Anaylsis of the reports show that the outbreaks for which no cause was found were largely those which for one reason or another were not thoroughly investigated. The number of sporadic cases notified has increased from 2,987 in 1950 to 6,534 in 1956, and information regarding the source of these cases is even more limited. [A further revision of this Memorandum is opportune.]

Notification

Section 26 (1) of the Food and Drugs Act, 1955, provides that if a registered medical practitioner becomes aware, or has reason to suspect, that any patient whom he is attending is suffering from food poisoning, he shall forthwith send to the medical officer of health of the district a certificate stating (*a*) the name, age and sex of the patient, and the address of the premises where the patient is; and (*b*) particulars of the patient's illness. Where the local authority of the district is not the local health authority, the medical officer of health is required to send a copy of the certificate within 12 hours after its receipt to the local health authority.

The main object of Section 26 is to ensure that the medical officer of health is informed of all illness occurring in his district and believed to have been caused by food. Although food poisoning is nowhere defined, the Minister does not consider that notification under Section 26 should include cases in which food has caused an infectious disease which is

separately notifiable in its own right. Attention is drawn to this point because where the disease has been food-borne some medical officers of health have customarily included notifications of such specific infections as paratyphoid fever and bacillary dysentery in their annual returns of food poisoning.

The Registrar General has requested medical officers of health to include notified cases of food poisoning in their weekly returns and also to make a quarterly return of the total of such cases amended by reason of corrected diagnosis.

Investigation of Notified Cases

This usually involves prompt, careful and at times persistent enquiries at the homes and other places connected with the affected persons as well as the collection of suitable material for laboratory examination. At the outset of the investigation it may be possible to prevent further cases by stopping the sale of suspected food and by recovering unconsumed portions already sold. Without attempting to prescribe the method of such enquiries guidance is given in Appendix A as to the chief headings under which investigation can best be made, and Appendix C describes in detail the considerations which apply to the collection of material for laboratory examination.

The Food and Drugs Act, 1955, does not give power to Local Authorities to order a temporary closing of food premises to prevent the sale of food which may be infected or may have become infected. Powers are available to stop the sale of suspected food and, under the Public Health (Infectious Diseases) Regulations, 1953, to require the exclusion from work of food handlers who are suffering from, or are known carriers of, certain food poisoning organisms. A timely reminder, where required, that a trader may be taking a risk in selling the food in question, may be found helpful.

Public Health Inspectors are competent to conduct many of the field enquiries. Such matters as the differentiation of the types of food poisoning which can often be made on clinical grounds (see Appendix B), and the collection of certain specimens from patients or suspected carriers of infection, require medical training and knowledge. In large outbreaks, where the amount of such work is likely to be beyond the medical resources of the Public Health Department concerned, assistance may be sought from the Ministry of Health and the nearest Laboratory of the Public Health Laboratory Service.

Because of discussions which the Ministry may need to have with the trade interests concerned, it would be helpful if a medical officer of health who asks the Public Health Laboratory to assist in enquiring into any outbreak would inform the Ministry at the same time that he has done so.

Enquiries on lines suggested by clinical or epidemiological evidence should never be held up pending the results of bacteriological or chemical examinations. Where the contamination, infection, or sale of the suspected food is thought to have occurred outside the district of the Medical Officer of Health, he should inform his colleagues in other districts concerned.

Collection of Data on Food Poisoning

Medical officers of health will undoubtedly wish that the greatest possible use should be made of information obtained by them during the investigation of cases of food poisoning. While the publication of such information in their annual reports and in articles contributed to medical journals is of considerable value, it is only by the collection and review of data on a national basis that some of the factors concerned in food poisoning are becoming apparent and their relative importance assessed. The suggested forms of report, which were included as Appendices D (i) and D (ii) to the 1949 revision, were devised to this end. D (i) has been used when analysing each year's total of notified cases of food poisoning and when rendering annual returns to the Ministry of Health, and D (ii) when summarizing the data of small or large outbreaks and when sending individual reports to the Ministry as soon as possible. These two forms have been revised and now make provision for reporting cases which come to the notice of the Medical Officer of Health otherwise than through notification under the Food and Drugs Act; both forms now also provide for a return of Salmonella infections which are not considered to be food-borne. The experience of the past seven years has shown that these two forms have been acceptable to Medical Officers of Health, many of whom continue to make the fullest use of these forms of report. Information extracted from both current reports and annual returns, collated with data available to the Public Health Laboratory Service, has made it possible to compile each year a fairly comprehensive report on food poisoning in England and Wales. An even wider use could be made of form D (ii) in sending a summary to the Ministry of Health as soon as convenient after each outbreak.* Reports on outbreaks as well as annual returns should be addressed to the Secretary, Ministry of Health, Savile Row, London, W.1.

Prevention of Food Poisoning

A careful compilation of the statistics of food poisoning is essential in any attempt to diminish the frequency of this condition. After making full allowance for better ascertainment, a study of the successive Annual Reports of the Chief Medical Officer of the Ministry of Health up to 1956 shows no evidence of any decline in the number of cases of food poisoning or in the proportion of large outbreaks. The Food Hygiene Regulations, 1955, which came into force on the 1 January 1956, lay down requirements concerning the handling of food, personnel, premises, equipment and restrictive temperatures applicable to certain foods. Investigations of outbreaks have, however, revealed some important factors in their causation and these might be listed as follows:

(i) The foodstuff, or one of its ingredients, may be primarily infected and the infection may survive the cooking or other preparation of the food.

* The specific duty of reporting to the Ministry any serious outbreak of notifiable disease and of sending to the Ministry a copy of any special report he may make to his Authority is laid on the Medical Officer of Health by Article 17 (6) and (7) of the Sanitary Officers (Outside London) Regulations, 1935, and in London by Article 14 (4) and (5) of the Sanitary Officers Order, 1926.

(ii) A primarily infected article may contaminate equipment and lead to secondary infection of other food products.

(iii) The amount of noxious material which survives cooking may be so small that no harm would result from immediate consumption. With delay in consumption, however, inappropriate storage, including misuse of refrigeration, and bad handling can lead to such growth of organisms, sometimes with the formation of enterotoxin, as will cause frank disease.

(iv) An infection introduced by food handlers can survive and multiply in such products as cream, imitation cream, custard and table sweets, cold meat, meat products, soups and gravies. These foods can easily become dangerous under certain conditions of domestic storage, though they would remain sound and comparatively free of risk if stored under ideal conditions in well-equipped premises.

Since the last revision of this Memorandum the Public Health (Infectious Diseases) Regulations, 1927, have been replaced by those of 1953. The Local Authority, or the Medical Officer of Health, in urgent cases if authorized by his authority, now has power to prohibit sufferers from, and known carriers of a recognised food poisoning infection from following any occupation connected with the handling of food if this involves a risk of spreading infection. The Regulations also require that facilities should be provided by an employer for the Medical Officer of Health to make a medical examination of an employee suspected of being a carrier of infection. The compensation provisions in Section 278 of the Public Health Act, 1936, are correspondingly extended to action taken under the Regulations. In practice, the Regulations have proved sufficiently wide and flexible to meet most contingencies, but it should be noted that there are no powers to suspend or to compensate a food handler who is merely a suspected carrier. The owner can however be warned of the possibility that if he employs a carrier on certain tasks the food may become infected and the gravity of the consequences that might result can be clearly explained to him.

APPENDIX A

Headings of Enquiry into Outbreaks of Food Poisoning

1. *Extent of Outbreak*

A complete list of patients (notified or otherwise), with ages and occupation, should be obtained by means of visits to affected households, institutions, etc., and by enquiries of medical practitioners in the area and of neighbouring medical officers of health.

2. *Clinical Features of Illness*

In each case a note should be made of the date and time of the first symptoms, the nature of the initial and subsequent symptoms, their severity, their duration, and whether accompanied by fever or followed

by the development of physical signs or lesions of the central nervous system.

3. *Evidence Implicating Particular Food*

A note should be made of:

(a) the date and hour at which suspected food was consumed by each affected person;
(b) names of persons at risk who consumed the suspected food but remained unaffected;
(c) names of persons who were taken ill at the same time but did not consume the suspected food.

4. *Identification of Agents Contaminating or Infecting Food*

Consideration should be given to:

(a) the type of agent which is suggested by the clinical features of the illness and the interval between consumption of the suspected food and onset of symptoms (see Appendix 'B');
(b) the nature of the suspected food as a medium for bacterial growth;
(c) the success or otherwise of attempts to isolate suspected bacterial or chemical agents from cases;
(d) the results of attempts to isolate an agent from samples of suspected food and its ingredients.

5. *Source and Means of Contamination of Food by Chemical or Bacterial Agent suspected*

Consideration should be given to:

(a) the epidemiology of the outbreak as evidenced in the grouping of cases, the distribution of the food, its origin and possibilities of contamination;
(b) the circumstances associated with the source, preparation, storage and distribution of the food and its constituent products;
(c) a history of previous or current illness of a significant kind in persons associated with preparation and distribution of suspected food;
(d) the results of clinical and laboratory investigation of persons associated with preparation and distribution of the suspected food.
(e) the nature of the food and its ingredients, and whether it was infected directly or indirectly by other contaminated foodstuffs or utensils.

APPENDIX B

(1) Clinical Features of the Illness Associated with Various Food Poisoning Agents

1. *Chemical Poisoning*

A characteristic feature is the shortness of the interval between ingestion and onset of symptoms—from 10 minutes to 2 hours. Initial symptoms are nausea and abdominal pain and in many cases there may be complaint of a metallic taste to the suspected food or drink.

Vomiting and diarrhoea may follow within half an hour, which may help the patient to get rid of the poison, but may also cause collapse in the elderly or the very young.

Chemical food poisoning is rare. Instances reported have included zinc poisoning from galvanized iron containers used for stewing or holding acid fruit, poisoning by metallic tin from sardines in tomato sauce (an institutional outbreak), lead poisoning from milk because the milk bottle had been previously used to hold white lead (a single case in a publican's wife), and poisoning by a heavy metal, probably lead, in a party of campers who had prepared stew in a utensil previously used to melt solder. Antimony poisoning has resulted when a drink containing citric or tartaric acid has been held in enamelware of an inferior quality.

Except when deliberately added with malicious intent arsenic in food is seldom encountered as a cause of gastro-intestinal symptoms of an acute type.

Diarrhoea and vomiting may follow the use of silver ware that has been cleaned with silver polish containing cyanide. The interval between ingestion and onset is considerably longer than in most kinds of chemical food poisoning, being usually 4 to 8 hours.

When an acute abdominal disturbance of food poisoning type is followed by muscular paresis, ingestion of sodium fluoride (a white powder which has been mistaken for baking powder) should be suspected. Ortho tri-cresyl phosphate (an oily fluid sometimes mistaken for edible oil) likewise gives rise to flaccid paralysis which may be preceded by symptoms of gastro-enteritis; paralysis, however, does not usually occur for 10 to 20 days after eating the contaminated food.

Contamination of food in bulk from spillage of an insecticide has been reported. Cases of food poisoning due to pesticides have resulted from accidental incorporation of these substances into food during its preparation; this is likely to occur if pesticides in white powder form are left in unlabelled containers in places where food is prepared. Poisoning by DDT is accompanied by nausea and vomiting with paraesthesiae and possibly twitching of the limbs. With BHC, *aldrin*, *dieldrin* or *endrin* violent convulsions, not necessarily fatal, may occur. With poisoning by *organo-phosphorus* insecticides sweating, nausea, colic and vomiting are the outstanding symptoms. A blood cholinesterase determination within 24 hours of the incident might help in establishing the diagnosis.

2. *Salmonella Infection*

The ingestion-onset interval is rarely shorter than 8 hours and may be as long as 72 hours. It is most often between 12 and 24 hours. The illness is a true infection and is not due to preformed toxins (see Staphylococci below). The onset is usually sudden, with abdominal pain, diarrhoea and frequent vomiting. Fever is nearly always present. There is a wide range in the severity and duration of the illness experienced by different individuals in the same outbreak. The length of the ingestion-onset interval and the degree of severity of the symptoms cannot be associated with the particular salmonella types. Anorexia and looseness of the

bowels often persist for several days. Cases which show clinical evidence of a blood stream invasion or of meningitis may prove fatal.

It should be borne in mind that the causal organism of paratyphoid fever is itself a salmonella, capable of causing an initial illness in no way distinguishable clinically from food poisoning. Such initial illness may later be followed by a further illness presenting the classical symptoms of paratyphoid fever. In some outbreaks associated with *Salm. paratyphi* it has been noted that those who suffered a marked initial gastro-enteritis escaped further illness, while others who escaped the initial gastro-intestinal upset later developed paratyphoid fever.

3. *Bacterial intoxications:* (*a*) *Staphylococci*

The ingestion-onset period is usually from 1 to 6 hours. Salivation, nausea, vomiting and abdominal pain are the chief symptoms but the associated diarrhoea and prostration with a subnormal temperature and lowered blood pressure are sometimes severe. The illness varies in duration probably according to the amount of the preformed toxin ingested and the susceptibility of the individual, but does not commonly last more than a day or two and is not fatal to healthy persons.

Where, as so frequently happens, nausea and vomiting are the presenting symptoms, confusion can readily arise between staphylococcal food poisoning and epidemic nausea and vomiting (*q.v.*).

(*b*) *Cl. botulinum*

Symptoms of botulism usually appear from 12–36 hours after ingestion of the toxin. Change of voice such as hoarseness is often the first sign noticed. Often lassitude or fatigue accompanied by dizziness or headache are first symptoms, occasionally nausea and vomiting. Soon ocular disturbances such as double vision become the chief complaint; later, difficulty in speaking and swallowing become prominent features and on examination there are ptosis and other signs of paralysis of the cranial nerves, especially the third and sixth, with loss of the light reflex and impaired function of the external rectus muscle. There is often abdominal distension but no pain and no fever unless pneumonia supervenes. Constipation is generally marked but there is no retention of urine. In fatal cases death commonly occurs from the third to the sixth day, usually from respiratory paralysis. The mentality remains clear up to a short time before death.

(*c*) *Cl. welchii*

The ingestion-onset interval is between 8 and 22 hours, usually 10 to 12 hours. It is not known with certainty whether the illness is due to the production of toxin derived from ingested clostridia or from pre-formed toxin. The onset is usually sudden with abdominal pain followed by diarrhoea. Nausea and vomiting are often absent; it is the abdominal pain and diarrhoea unaccompanied by sickness which are so characteristic of this form of food poisoning. The illness is of short duration, one day or less, and is not fatal in healthy persons.

4. Other Bacteria

In some outbreaks of food poisoning the organisms mentioned in 2 and 3 above cannot be detected. Examination may reveal gross contamination with organisms which are usually disregarded if present in small numbers. From time to time outbreaks have been reported in which the causal agents have been α-haemolytic streptococci, aerobic sporing bacilli and even *Proteus* and coliform bacteria. It seems probable that these and similar 'non-specific' organisms when allowed to proliferate in certain foodstuffs are able to produce toxic substances.

In these outbreaks the interval between ingestion and onset varies from 3 hours up to 18 hours or more. The symptoms vary from those simulating salmonella food poisoning to those resembling food poisoning from toxin. Paracolon bacteria, for example, may cause symptoms resembling the first type. On the other hand *Bacillus cereus*, an aerobic spore-bearing bacillus, and α-haemolytic steptococci may cause symptoms resembling the second type, namely pain and diarrhoea, or vomiting and diarrhoea.

The foods concerned are those usually favourable to bacterial growth, and frequently cooked a day or more before required, e.g. made-up meat dishes, gravy, trifles, custards and purees.

The principal features of the several types of food poisoning are summarized below, in the order of approximate incubation period.

Agent	*Ingestion/onset*	*Main symptoms*
Chemical (irritant)	Short—10 minutes to 2 hours	Nausea, abdominal pain, then vomiting and diarrhoea.
Staphylococci	1–6 hours	Salivation, nausea, vomiting, abdominal pain, prostration and subnormal temperature.
Salmonellae	12–24 hours	Abdominal pain, diarrhoea, vomiting and fever.
Cl. welchii	8–22 hours	Abdominal pain and diarrhoea.
'Non-specific' bacteria	3–18 hours	Diarrhoea, abdominal pain, vomiting.
Cl. botulinum	12–36 hours	Change of voice, diplopia, ptosis. Cranial nerve palsies. Obstinate constipation.
Chemical (neurotoxic)	(i) Early (ii) 10–12 *days*	(i) Early muscular paresis (e.g. sodium fluoride). (ii) Delayed flaccid paralysis (e.g. ortho tri-cresyl phosphate, an oily fluid, sometimes mistaken for edible oil)

(2) Differential Diagnosis of Food Poisoning from other Communicable Diseases

Nausea, vomiting, diarrhoea and abdominal pain are the chief clinical features of several common communicable diseases as well as of food poisoning. The chief diseases which, when they occur in the form of localized outbreaks, may be mistaken for food poisoning are:

(*a*) *Bacillary dysentery*

A disease of acute onset with diarrhoea as its chief symptom and often associated with fever and tenesmus. In severe cases the frequent stools contain blood and mucus. In minor cases the symptoms other than diarrhoea vary and recognition may be difficult except by means of laboratory examination. The disease is caused by various species of the genus *Shigella* (*Flexner*, *Shiga*, *Sonne*), which can readily be isolated from the faeces in the acute stage. Outbreaks are usually associated with institutions such as mental hospitals, and children's nurseries, and may at times be caused by contaminated food, but are more commonly due to cross-infection.

(*b*) *Epidemic Sore Throat*

When this disease is caused by raw milk infected with haemolytic streptococci, vomiting is a frequent early sign. The isolation of Group 'A' streptococci from the fauces readily enables this illness to be distinguished from food poisoning.

(*c*) *Epidemic Nausea and Vomiting*

This appears to be a separate entity a group of diseases of unknown origin commonly called 'Gastric flu'. The symptoms are chiefly nausea with irritation of the upper alimentary tract but an associated diarrhoea is not uncommon. Fever above 100°F. is unusual. The incubation period is from 2 to 7 days and in an affected household or community there may be an interval of days between cases or groups of cases (cf. the common cold). Although food poisoning may be suspected it can usually be excluded on circumstantial grounds as well as by the completely negative results of the laboratory examination of all specimens and samples. It is possible that a virus (or a group of viruses) is the cause of this condition and that the modes of transmission resemble those of influenza.

This illness is often known as winter vomiting disease, though its incidence is not confined to the winter months, nor is vomiting quite as prominent a symptom as nausea. Since the previous revision of this Memorandum certain work in the United States of America has given added support to the belief that some forms of this condition may be due to a filter-passing agent of intestinal origin.

APPENDIX C

Field and Laboratory Investigation

Speed is essential in the investigation and control of an outbreak of food poisoning, and the first objective is to prevent the consumption of more of the infected aliment. The longer the period between ingestion

of the food and onset of signs and symptoms the more difficult it may be to ascertain the true cause of the outbreak; for this reason prompt notification of the case is most important. A carefully-taken history of the circumstances of the outbreak and of the illnesses of the patients is well repaid and enables effort to be directed to the proper quarter. Medical practitioners should be reminded that when food poisoning is first suspected steps should immediately be taken to ensure that no left-over food or vomit is thrown away. On becoming aware of an outbreak of food poisoning the medical officer of health should forthwith warn the nearest public health laboratory to expect the arrival of specimens for examination. A Public Health Inspector sent to begin the investigation of an outbreak should concentrate on preventing any further consumption of suspected foods and arranging for the collection and conveyance to the laboratory of samples of these foods as well as specimens of faeces and vomit from the persons affected. It should be borne in mind that suspected food which may already have been sold may not necessarily have been consumed, and suitable warning to customers can frequently be given by telephone, visit, or mobile loudspeaker van, or where there is no alternative through the British Broadcasting Corporation's system.

Enquiries carried out along the lines suggested in Appendix A should narrow down suspicion to one or two particular articles of food. Any left over remnants of these foods, however small, should be taken at once to the public health laboratory.

In outbreaks of bacterial food poisoning the foods most likely to cause trouble are cooked meat (whether produced in the home or in the factory), gravy, eggs, milk products and imitation cream. There is particular risk when susceptible foodstuffs have been cooked, allowed to cool slowly, and then re-heated prior to consumption. Foods with a high sugar content (greater than 40 per cent), acid foods (pH less than 4·0) and fats are not usually associated with bacterial food poisoning though they may be with chemical food poisoning.

When canned foods are suspected the remains of the particular can should be submitted for examination. Dried foods should not be excluded as possible sources of food poisoning. Code numbers, markings and other relevant details should be recorded so that investigations can be made into the conditions of manufacture and preparation, if and when necessary. Such information is also required for laboratory records. In following up incriminated foodstuffs it is often necessary for Medical Officers of Health to ascertain the name or names of suppliers, distributors, and importers concerned, the country of origin and, in some cases, details of the particular shipment.

Sterile containers and equipment for sampling are usually available from the laboratory and advice on methods of sampling will be given by laboratory directors when necessary.

The following quantities of material should be sent to laboratories:

Imitation cream: at least 50 gm. (2 to 3 oz.).

Cakes filled with imitation cream: sufficient to provide at least 50 gm. of cream.

Dried foods: at least 50 gm. of each sample, in sterile containers.

In addition information should be given on the method of preparation of foods, how stored between preparation and consumption, and for how long.

Within 24 hours of receiving material the laboratory should be able to give a preliminary report that will help to direct further investigation to tracing the source of contamination or infection and defining the conditions that contributed to the occurrence of the outbreak.

Chemical Poisoning

Where chemical poisoning is suspected, it is most important to secure specimens of the vomit as well as of the suspected food. Should any of the cases prove fatal, the stomach and its contents, together with a portion of the liver, should be reserved for examination.

Search for the mode of contamination of the food with chemical substances may necessitate the examination of packages, bags, or containers from which the food has been taken (e.g. for evidence of staining with a chemical agent such as arsenical weed-killer; for traces of tar oil in barrels and drums subsequently used for edible oils). Samples of all suspected articles should be obtained for laboratory investigation. It may also be important to examine in the laboratory cooking utensils such as galvanized iron pans in which acid fruits have been boiled or powders whose identity is in doubt, and unusual types of cleaning material or polish that have been used. All such samples and articles should first be taken to the public health laboratory, and the bacteriologist there, after excluding the likelihood of bacterial food poisoning, will pass them on to a Public Analyst, for chemical examination.

Food poisoning due to salmonella infection

Salmonella food poisoning is due to a true infection of the gastro-intestinal tract, and the most frequent interval between ingestion of the food concerned and onset of symptoms is about 18 hours. Although causative organisms can only rarely be found in the vomit they can usually be isolated from the faeces during the first 2 or 3 days of the patient's illness. Subsequently this is more difficult although several careful studies have shown that some of those infected remain excreters for weeks or months.

When salmonella infection proves fatal, and if a diagnosis has not already been made, post-mortem specimens of ileum, spleen and liver should be submitted to the laboratory. A majority of the deaths from salmonella infection occur among infants, the aged and those debilitated by some chronic disease.

It should be remembered that some organisms of the salmonella group find their natural habitat in such domestic animals as cattle, pigs and poultry, and others in such vermin as rats and mice. Evidence has come forward in recent years that domestic pets—cats and dogs—can be salmonella carriers. Human food poisoning may be caused by consuming beef, pork or offal which have been derived from animals with a septicaemic salmonella infection or have been contaminated in

the slaughter-house by infected intestinal contents; milk from infected cows may contain secreted salmonella or be contaminated by the animals' faeces. Eggs, more especially from the duck, whose eliminatory or genito-urinary tract is liable to be infected, may spread salmonella, and this hazard includes dried and frozen eggs prepared from infected pulp. Food may be contaminated by the faeces of salmonella-infected rats or mice.

It has recently been shown experimentally that cooking does not always destroy all the salmonellae present in meat pies to which known amounts of a standarized culture had been added prior to baking.* There is in fact only a narrow margin of safety in many of the accepted practices of commercial baking and a slight departure from these can permit the survival of sufficient micro-organisms to multiply rapidly to pathogenic numbers.

Outbreaks of salmonella infection are sometimes caused by transient human excreters who may contaminate food directly or indirectly when employed in its preparation. Made-up meat dishes, gelatine solution (commonly used in the manufacture of meat pies and other meat products), custards, trifles and sweets garnished with imitation cream are particularly favourable to bacterial multiplication; they may become highly infected, especially after being left a few hours at a warm temperature. When an outbreak caused in this way has occurred, interpretation of the bacteriological findings in the workers may become particularly difficult because, by the time the infecting organism has been isolated and a bacteriological search has been made among the staff, the individual who contaminated the food may have ceased to be a carrier, or may have partaken of the infected food, while on the other hand there may be others who are found to be infected because they have eaten the infected food. Recent experience suggests that human excreters may not be so important as originators of outbreaks as is the introduction of contaminated food or ingredients. Discovery of one or more infected members of the staff should not, therefore, be accepted as the explanation of an outbreak until all possibility of an extra-human source can be excluded.

The investigation of outbreaks of salmonella food poisoning often requires the most painstaking enquiry guided by extensive knowledge and experience of potential sources of infection. The food that caused an outbreak has often been completely consumed or thrown away by the time the enquiry is made, and the tests which can then be done therefore afford only circumstantial evidence of the mode of infection.

When the suspected food is no longer available and other food from the same batch or consignment cannot be obtained, attention should be directed towards the most probable sources of contamination of the food. Investigations may have to be extended to slaughterhouses, farms, warehouses and other premises. The examination of food handlers for evidence of infection is advisable. If difficulty is experienced in persuading all members of the kitchen staff to provide faecal specimens, it should be explained that the object is not to cast blame on anyone but to make certain that recurrence from a human source will be prevented.

* Cmd. Rpt. No. 96, H.M.S.O., 1955.

Sporadic single cases constitute a considerable proportion of all reported salmonella infections and deserve much more attention than they usually receive. Full information is needed about the source of infection of these apparently isolated cases, which may themselves act as reservoirs of infection. Only careful epidemiological enquiries accompanied by exact serological identification of the infecting organisms will provide this knowledge. These enquiries should be made by medical staff working in close collaboration with the bacteriologist. The problem offers an interesting and important field of study and should, if successfully elucidated, greatly enlarge our knowledge of the sources and mode of spread of salmonella infections.

Medical officers are reminded that 'salmonellosis' is not a notifiable disease under the Public Health Act, 1936, and food poisoning notifiable under the Food and Drugs Act, 1955, does not of course include all forms of salmonella infection. Where illness due to salmonellae has spread by cross-infection or by case-to-case contact, and food is concerned only indirectly or not at all, food poisoning may at first have been suspected and the case notified. These notifications should be corrected, but the cases reported with any other cases of salmonellosis that are not statutorily notifiable. Provision has been made in the revision of Appendix D (i) and (ii) for the separate return of salmonella infections which are not considered to be food-borne.

Bacterial intoxications: (a) Staphylococcal toxin food poisoning

Where the symptoms and the interval between ingestion and onset of illness suggest poisoning with staphylococcal enterotoxin, primary attention should be directed to the food handlers, e.g. kitchen staff. A clinical examination should be made for evidence of septic lesions of the hands, arms or face, a purulent discharge from the nose or ear, or obvious abnormality of the upper respiratory tract, and persons suffering from any of these conditions should be at once excluded from work.

Swabs should be taken from the local lesions of those found to be suffering from sepsis, as well as from the anterior nares and hands (including the backs of the hands) of *all* food handlers.

Specimens of food, and of vomit and faeces from the patients, should be submitted for bacteriological examination.

The isolation of coagulase-positive staphylococci from any of the material examined is not in itself sufficient to warrant the conclusion that the person from whom it came is the cause of an outbreak. About 40 per cent of adults carry such organisms in the nose, and 10–20 per cent on their hands. Any strains isolated should be submitted to a reference laboratory for serological or bacteriophage typing. A presumptive case can be made out against the food handlers if strains isolated from them are of the same type as those recovered from the food and/or the patients.

In outbreaks due to milk or milk products the staphylococci are often derived from cows suffering from mastitis in the dairy herd concerned.

The final step would be to prove by feeding experiments that the organisms isolated are capable of producing enterotoxin. Unfortunately

no laboratory animal is really satisfactory for this purpose, and unless human volunteers can be obtained this cannot be done.

In the rare instances in which staphylococcal food poisoning proves fatal, the stomach with its contents and the upper part of the jejunum should be reserved for bacteriological examination. Very few records are available of the post-mortem appearances in this type of toxaemia and particulars of any carefully observed cases should be sent to the Ministry of Health.

(b) Food poisoning due to Cl. welchii

α- or non-haemolytic heat-resistant strains of type A *Clostridium welchii* are frequently found in association with outbreaks of food poisoning. The vehicle of infection is almost invariably meat which has been boiled, steamed, braised, stewed, or insufficiently roasted, allowed to cool slowly and then eaten either cold or re-heated on the following day. Gravy or stock infected from the meat, and not freshly prepared, is another hazard.

Between 2 and 5 per cent of the population has been found to excrete *Cl. welchii*, and heat resistant strains of this organism have been found in as many as 24 per cent of a series of beef carcases from a slaughterhouse. The discovery of a carrier among the staff engaged in preparing food does not necessarily mean therefore that he was the source of the infection as the organism might well have been on the meat when it arrived in the kitchen.

It will be apparent that the prevention of this form of food poisoning is more dependent on the control of cooking technique and storage than on an elaborate search for and elimination of carriers.

Food poisoning due to other bacteria

Some outbreaks of food poisoning occur in which laboratory examination gives no evidence of staphylococcal, salmonella or *Cl. welchii* infection. In many of these outbreaks the suspected food is one favourable to bacterial multiplication and culture in the laboratory shows the presence of enormous numbers of micro-organisms that are not normally pathogenic by mouth. As a rule one type of organism of this kind such as a paracolon bacterium, an α-haemolytic streptococcus, a member of the proteus group, or one of the aerobic or anaerobic spore bearers is predominant.

Except by using human volunteers for feeding experiments, it is impossible to prove that any one particular organism is a cause of such outbreaks. The few experiments of this kind that have been made together with other evidence leave no doubt that some of these organisms when present in the food in large numbers may cause vomiting and diarrhoea; as already mentioned in Appendix B, illness caused in this way usually occurs about 3–18 hours after ingestion. Symptoms are seldom severe, and recovery is ordinarily rapid.

Most of these outbreaks have followed the eating of food prepared in kitchens attached to communal establishments, such as schools and canteens. Particularly suspect in this connection are articles of food such as stews, gravies, custards and trifles, which have been prepared

on the day before the meal and have been allowed to cool slowly at atmospheric temperature, or even, if the food is in large containers, in the refrigerator overnight. Bacterial multiplication can occur very readily in such foods and samples for examination in the laboratory should be taken at the earliest possible moment. Unless the samples can reach the laboratory within an hour they should first be cooled down and then well packed in ice. When food residues have been kept in a warm place before being sampled it may be impossible to form an opinion on the degree of bacterial contamination at the time the food was eaten.

Outbreaks of this sort are particularly liable to occur under unhygienic conditions of food preparation, and the standard of kitchen hygiene should be investigated in all cases.

APPENDIX D (i)

(see page 385)

Annual Return of Food Poisoning

(Salmonella Infections that are not considered to be food borne should not be included under items (2), (3) *or* (4), *but should be shown separately under item* (5))

1. Local Authority   Year

2.—(*a*) Food Poisoning Notifications (Corrected) as Returned to Registrar General

1st Quarter   *2nd Quarter*   *3rd Quarter*   *4th Quarter*   *TOTAL*

(*b*) Cases Otherwise Ascertained

1st Quarter   *2nd Quarter*   *3rd Quarter*   *4th Quarter*   *TOTAL*

Note: Symptomless excreters should not be regarded as cases, and any notification of a symptomless excreter should be corrected. At the same time the numbers of symptomless excreters may at the Authority's discretion be entered here, separately.

(*c*) Fatal Cases

1st Quarter   *2nd Quarter*   *3rd Quarter*   *4th Quarter*   *TOTAL*

3. Particulars of Outbreaks

Details of each outbreak to be given as in Appendix D (ii).

	No. of outbreaks		*No. of cases*		*Total No. of cases*
	Family outbreaks	*Other outbreaks*	*Notified*	*Otherwise ascertained*	
Agent identified*					
Agent not identified					

4. Single Cases

	No. of cases		*Total No. of cases*
	Notified	*Otherwise ascertained*	
Agent identified*			
Agent not identified			

*To be classified according to agents.

(*a*) Chemical Poisons (type to be stated).
(*b*) Salmonella (type to be stated).
(*c*) Staphylococci (including toxin).
(*d*) *Cl. botulinum.*
(*e*) *Cl. welchii*
(*f*) Other bacteria (to be named).

5. Salmonella Infections, not Food-Borne

Salmonella (type)	*Outbreaks*		*No. of cases*	*Single cases*	*Total No. of cases (Outbreaks and single cases)*
	Family	*Other*	*(outbreaks)*		

APPENDIX D (ii)

(See page 385)

Report on Individual Food Poisoning Outbreak (Summary of details)

(To be sent to Ministry of Health as soon as the Information is available)

1. Food Causing Outbreak. Agent Causing Outbreak.

2. Cases Forming Outbreak which occurred from to
   Notified *Otherwise ascertained* *TOTAL* *Fatal*

3. Clinical Features. Average interval ingestion to onset (hours)—
   Main symptoms
   Severity of illness *Duration of illness*

4. Results of Laboratory Investigation (Summary)
   Cases *Food handlers*
   Food samples *Other*

5. Origin and Preparation of Food Causing Illness.

6. Place at which Food Causing Illness was Consumed.
   Estimated number of consumers at risk.

7. Probable Origin of Infection or Contamination of Food.
   Contributory Factors.

Notes

1. Where canned, packaged or imported goods are implicated, full information should be given of brand, other markings and supplier.

Agents causing outbreaks when identified should be stated, i.e., chemical poison, type of salmonella organism, staphylococci, etc.

6. Place at which food causing illness was consumed should be stated, i.e., home, school, public restaurant, canteen or railway restaurant car, etc.

7. Contributory factors such as poor food storage facilities, lack of refrigeration should be stated if known.

Where salmonella infection is found not to have been food borne Appendix D (ii) may be used when reporting the circumstances to the Ministry, with the appropriate amendments to the above headings.

APPENDIX II

Gastro-Enteritis Aboard Ship

Control of Outbreaks

When an outbreak of gastro-enteritis occurs aboard ship, the cause should be established and brought under control as soon as possible. This can usually be accomplished by taking a history of each case. The use of previously prepared 4 × 5 cards for this purpose is recommended. The important points to be noted are: time of onset, symptoms, division of ship's company to which attached, and food eaten (all food eaten within the past 24 hours, or at least which meals were eaten in the previous 48 hours). It may be necessary to get a more detailed history when the cause is narrowed to one or two meals or food.

The symptoms, incubation time, etc., should suggest a diagnosis of (1) chemical or staphylococcal toxin food poisoning, or (2) food infection due to *Shigella* or *Salmonella*. The incubation time of the first group is usually ½ to 5 hours, and of the second, usually from 12 to 72 hours. Those ill due to the first group (food poisoning) tend to have more nausea and vomiting, but less fever, than those ill due to food infection. Outbreaks due to a 'virus' infection are not explosive in character. When the responsible food is found, its preparation and handling should be carefully checked to determine the mechanisms of the outbreaks.

A diagnosis of the type of infection or poisoning should be made, if possible, because control procedures differ, depending on the etiology. Food-poisoning outbreaks (staphylococcic enterotoxin or chemical) are most often self-limited. When the cause of the poisoning has been determined, measures to prevent continuance or recurrence should be instituted. Gastro-enteritis caused by food infection is frequently not self-limited, and active control measures must be taken immediately. Food-handler carriers are the most frequent source of infection. This being the case, one carrier may start an epidemic, infecting more food handlers who may become carriers either as active clinical cases or as asymptomatic cases. Experience has shown that from 10 to 35 per cent of asymptomatic food handlers have rectal cultures positive for *Shigella* organisms as long as 2 months after the original outbreak. *Salmonella* were not found in as high a percentage of food handlers but many of these asymptomatic food handlers had a positive culture 1 month or more after the onset of the original outbreak.

If facilities for culturing stools are not available, all food handlers may be given sulfadiazine, and any among them with gastro-intestinal symptoms should be relieved of duty associated with the preparation and serving of food. Food handlers should be instructed in all the rules and methods of galley sanitation and personal hygiene, and these procedures should be enforced. Food-handler personnel should be inspected daily for signs and symptoms of illness.

Scrub brushes for the hands should be provided and a bucket of a disinfectant (lysol or cresol solution) should be placed at the door of

each head (latrine). A guard should be posted to see that everyone thoroughly washes his hands and dips them in the bucket before leaving. This guard can also be of great help in making all men who have diarrhoea report to the sick bay. The guard on watch in the head used by the food handlers should be particularly reliable. In a widespread outbreak of bacillary dysentery aboard many ships of the Third Fleet which were investigated, many of the cases were undoubtedly due to direct or indirect contact with patients.

If stool cultures of patients are negative for enteric pathogens, and the outbreak is evidently not due to a chemical poison or staphylococcal toxin, a virus should be suspected. In such cases, the possibility of contact transmission, as in respiratory disease, should also be considered.

Drinking water should always be checked bacteriologically if possible, even though it has been found to be an unusual source of infection.

Summary

In summary, the steps to be taken in case of an outbreak of gastroenteritis are as follows:

1. Obtain a history of each case as outlined above, and isolate the patient if possible.

Inspect the galley as outlined above, and determine the technique used in preparing all foods under suspicion. Obtain a record of menus as actually served during the previous 3 days. Having a daily 'field day' in all food-handling compartments as long as the epidemic lasts.

3. Obtain samples of suspected foods.

4. Examine all food handlers daily for skin lesions, and signs and symptoms of present or recent illness. Use a complete and up-to-date check-off list to be sure all handlers are examined. Remove all questionable men from this duty.

5. Give a short talk to all hands regarding the importance of personal hygiene and the modes of transmitting enteric diseases including transmission by carriers. The food handlers should be given special additional instructions.

6. Arrange for a 24-hour 'head watch' and employ hand brushes and disinfectant as outlined previously.

7. Check on the use of contaminated salt water on decks and anywhere in spaces used for food preparations and handling.

8. Particularly warn all hands and boat crews of the danger of contamination with polluted sea water while in and around the boats.

9. Give all food handlers prophylactic sulfadiazine, 1 gram 3 times a day for 5 days, if stool cultures cannot be made. Check on the source and handling of milk supply.

10. Chlorinate all fresh-water tanks to a strength of two parts per million. Investigate the source of the water supply. Check for the possibility of cross connections with salt-water lines, etc.

11. To prevent spread, do not transfer men ashore or to other ships except for treatment.

12. Report outbreak to line and medical officers (and to other higher authority if indicated) as directed, as soon as possible.

13. Obtain the assistance of an Epidemiological Unit, if available.

14. If a virus is suspected as the etiological agent, measures for the control of respiratory illnesses should also be instituted as follows:

(*a*) Isolate all patients if possible.

(*b*) Ascertain the habits and duties of the patients who first show the illness, and determine the locations aboard ship (division, bunking compartment, etc.) or special group, etc., in which the illness may have originated, and in which new cases may be expected to occur.

(*c*) Inspect the ship as outlined above for insanitary and unhygienic conditions, and have them corrected.

(*d*) Check 'head to foot' bunking arrangements and ventilation of bunking spaces.

(*e*) Check particularly the health of milk handlers and the methods used to mix and refrigerate milk and ice-cream.

(*f*) Give a short talk to all hands on the prevention of respiratory illnesses.

(*g*) Do not transfer men ashore or to other ships unless necessary.

If the service of an Epidemiological Unit is available during or after an outbreak of any kind, it is the duty of the medical officer to request an Epidemiological Unit survey as soon as possible. It is also advisable to ask for a routine epidemiological survey every 6 months.

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SUBJECT INDEX

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